

ON CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS

IN INFANTS.

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by

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I N T R O D U C T I O N .

It is now close on seventy years since Williamson of Leith⁵ recorded a "case of Scirrhus in the stomach probably congenital" ; which, in the light of more advanced knowledge, is believed really to have been an example of that condition, now most usually termed Congenital Hypertrophic Stenosis of the Pylorus. In other words, it is believed to be practically the first case to have been recorded in Medical Literature, with the exception perhaps of that described by Beardsley in the year 1788 (Osler

In Williamson's case, although the Pylorus appeared hard and indurated, it was found that the mucous and, even more, the submucous coats were mainly responsible for this increased thickness of the Pyloric wall; and to quote his own words, — there was present " a peculiar Hypertrophy or modification of the cellular tissues". When one considers this pathological record and compares it with those which eventually followed, one is at once struck by the fact that Williamson found an hypertrophy of submucous and/

and mucous coats, whereas, the great majority of his successors thought that in their cases this was negligible, and laid most stress on the hypertrophy that they found in the muscular coat. One, therefore, wonders whether his conclusions may have been somewhat erroneous, drawn perhaps from the examination of a faulty section; and, indeed, it could hardly be expected that pathological technique, in his time, should be so perfect as it became later on.

On the other hand, the pathology in his case may perhaps have been a little unusual. For instance, it may have been that there was ~~an~~ hypertrophy of the mucous coats almost great enough to occlude the lumen of the Pylorus, and that the Stenosis was finally completed by a condition of spasm of the Pyloric musculature.

A similar case was recorded by Dawosky⁵ in the following year, and was diagnosed by him, somewhat vaguely, as an Hypertrophy with induration of the submucous tissue.

Looking back into the past, one wonders why it was that a period of forty-six years then elapsed without any reference to this condition in the literature of medicine.

It could not have been that cases did not occur; one must then conclude that sufficient interest had not been aroused at the time the first one was recorded/

recorded, and the pioneer cases had, therefore, had been allowed to sink rather into oblivion. When Hirschsprung, in 1888, inaugurated the present new series of records, his doing so marked an epoch, since when literature has become rich in similar interesting cases, all of which have helped to place it, as a pathological entity, on a more secure footing in medical science.

Synopsis of Classical Cases.

Osler has pointed out that, in 1788, Hezekiah Beardsley, recorded a case of Congenital Hypertrophic Stenosis, but it was under the title of "Scirrhus of the Pylorus". This name possibly suggested itself on account of some resemblance of the enlarged Pylorus to a Neoplasm. It is to him, therefore, that the credit is due of having been the first to describe the pathology of this condition; even though he may not have realised, any more than those who immediately followed him, that this was to prove an all important discovery to the generations to come. It was generally believed that, in 1841, Williamson had recorded the first case of Pyloric Stenosis, until Osler⁵ corrected the mistake and proved that Beardsley had described this condition more than half a century earlier.

The third case was described by Dawosky⁵ in 1842, and then came the long period of forty six years /

years, during which no mention was made in medical literature of any similar cases, and the condition seems to have been almost forgotten. A new interest in it, however, was aroused by Hirschsprung⁵ in 1888, when he described two cases that had come under his care. Three years before this Maier⁵ had written on the subject describing 31 cases, mainly in adults, where a condition of Pyloric Stenosis had been found post mortem. Some of these cases had been examined by Maier himself, and a certain proportion by Landerer⁵. The age of the subjects, at the time of death, varied from 12-80 years, and Maier and Landerer held that, as no signs of ulceration nor of any other pathological change was found after death, that the Stenosis in these cases must, therefore, have been congenital.

This assumption led to a certain amount of controversy. As it was not possible for these two observers to prove that in their cases the condition had not been acquired in later life, their theory, that stenosis had existed from birth, fell rather into disrepute. The question, there arose as to whether such a stenosis ever did occur congenitally. It seems strange that the cases of Williamson and Dawosky should have been so easily forgotten, but the opportunity now arose for their pioneer work to be justified, when Hirschsprung therefore, in 1888, described a case of Pyloric Stenosis and Hypertrophy, occurring in/

in a child of only 30 days old, proof seemed at last established that this condition, may, indeed, be present from birth. As so often happens, once this conclusive evidence had been published and the symptoms and signs of Hirschsprung's two cases had been described, other observers were quick to follow, and from this time onwards literature has accumulated the description of a gradually increasing number of similar cases.

Peden⁵, in describing a case in the following year, noted that the three previous children in this family were all "great vomiters", apparently concluding that there was perhaps a family tendency, if not actually an hereditary influence affecting the etiology of this condition. Henschel⁵ too, in 1891 described four cases occurring in one family; but, as he himself says that there was present only a partial narrowing of the Pylorus, and does not appear to have been impressed by any very marked hypertrophy in any of them, it is quite possible that in these cases there may have been present ^{only} a slight hyperplasia, with a superadded spasm of the Pylorus causing symptoms of stenosis.

It will be noticed, therefore, that the earlier writers laid stress on the influence of heredity; whereas, now it is usually looked on as a very minor factor in the causation of congenital Hypertrophic Stenosis of the Pylorus, and this only in a very obscure/

obscure manner.

The records show that in 1891, a case was described by Newton Pitt.

Five years later in 1896, Dr John Thomson²⁸ described his first two cases, and emaciated his theory that the increase of muscle must have been the result of over-action, which must have occurred in utero, as it had evidently been going on for a long time. This exaggerated functional activity, he thought, might have been occasioned by a derangement (probably from faulty development) of the nervous mechanism which regulates the contraction and relaxation of the Pylorus under appropriate stimuli. His hypothesis, which has become so well known, has been carefully elaborated upon by him since then, and has given rise to endless speculation from all sides.

Three cases were described by Gran⁵ of Christiania in 1896, and one, also, by Findelstein⁵. It is interesting to note that this case of Findelstein's was diagnosed by him intra vitam after he had had the opportunity of seeing some of Gran's cases in Heubner's clinic.

In the same year De Bruyn Kops⁵ described a case and Schwyzer⁵ witnessed a Post-mortem which enabled him to diagnose during life, his second case of, what was considered then, a very rare condition./

condition.

The third case to be seen by Dr John Thomson²⁸ was in the year 1897. Aided now by the experience obtained from his two previous cases, he was able to make a correct diagnosis of the condition during life, which he later confirmed by a Post Mortem examination.

Four cases in all, were recorded during the following year : two by Ashby, one by Soltan Fenwick, and one by Thomas⁵. The specimen obtained from the last named case is now preserved in the Museum of St Bartholomew's Hospital: but, Cautley remarks that no specimen, dating from these early days has yet been found in the Museum of the Royal College of Surgeons.

In 1898, an exhaustive article by Meltzer¹⁶ on Congenital Pyloric Stenosis appeared in the Medical Record of that year. After briefly reviewing what had already been written on the subject, he went on to describe a case which he had diagnosed, and on which he had operated. This child was at first treated medically by dieting and gastric lavage, during which time Meltzer made some very interesting experiments. These gave valuable information as to the condition of the stomach at the time, and led him, theoretically, to divide up the life history of such a stomach, into 3 phases :- (1) Simple Insufficiency, (2) Attempted Compensation, (3) Atony or permanent Gastro-ectasy.

One/

One cannot but regret that his hopes of success after the operation were doomed to disappointment, owing merely to a fault in the surgical technique.

Cautley⁵, in 1899, described two cases typical of this condition and after dwelling on the symptoms and signs on which the diagnosis should be based, discussed the various operations then in vogue for overcoming the obstruction at the Pylorus.

The treatment of this interesting, and by no means rare, condition is still a very difficult problem, and it is instructive to notice the different phases through which it has moved. Here, as in most branches of medicine, one notices the rhythmic swing of the pendulum; now to one extreme, now to the other. Originally the treatment was purely medical and consisted of very careful dieting, aided later on by gastric lavage ~~among~~. About the time that Meltzer¹⁶ writes surgical treatment was apparently beginning to find favour, dating from Schwyzer's decision to employ Joreta's operation on one of his cases. Treatment by operation eventually became so popular that it was employed in almost every case as soon as the diagnosis had been definitely established. The pendulum has reached the full extent of its swing, and who can deny that it may swing back a little?

One cannot help feeling that the time will come for/

for a compromise ——— when it will be realised that certain genuine cases of Congenital Hypertrophic Stenosis of the Pylorus are suitable for operation, and that certain other equally genuine cases of this condition are not likely to benefit much by surgical treatment. The question naturally arises as to which cases are suitable for expectant treatment, and which are those in which an operation is indicated. This problem, unfortunately, appears to be ^{no} nearer a solution than it was a decade ago; and one, still, can only speculate as to the ^{prognosis} ~~progress~~ and most suitable treatment as each individual case presents itself. One's own feeling in the matter is perhaps somewhat conservative. It always seems a wise policy however, in combating disease, to assist nature as far as possible. This may be done in the case of Congenital Hypertrophic Stenosis of the Pylorus, tentatively any way, by carefully regulating the diet, and by instituting as a routine Gastric Lavage, once or twice daily, depending on the severity of the symptoms. By weighing the child every second day it is possible to know exactly how much or how little a conservative form of treatment is benefiting the particular case. Should there be no abatement of symptoms after a few days trial, or should any serious loss of weight occur, then no time should be lost in deciding to hand the case over to the Surgeon./

Surgeon. Should time be lost at this point, after it has once been decided that for this particular child ~~child~~ conservative treatment is useless, then the chances of recovery are appreciably lessened and the Surgeon's task thus made infinitely more difficult and hazardous.

Of all diseases in children this is probably the one, where it is most essential that the physician and the surgeon should work hand in hand. It is necessary that they should understand each other, and each understand his own limitations; and thus by skilful co-operation hope eventually to gain the mastery of this baffling condition.

E T I O L O G Y.

Occurrence : There is no doubt that the true form of Congenital Hypertrophic Stenosis of the Pylorus is still a comparatively rare condition, although it appears to be becoming commoner. This, however, is probably only because fewer cases are missed now that the doctor knows what definite symptoms and signs to look for. Some idea of its rarity may be gained by the fact that 5,312 was the total number of admissions to the medical wards of this hospital during the years 1916 - 1920 (inclusive), and that out/

out of that number there were only 22 cases of Congenital Pyloric Stenosis; which represents 0.41% of the total admissions. Further, it is only fair to point out that a hospital always tends to accumulate a deceptively large number of rare conditions, giving one the false idea that such cases are comparatively common. It is possible, therefore, that we have had a proportionately large share of Pyloric cases, and that 0.41 is a larger percentage than usually exists.

Social Scale : This is one of the few diseases which appears to affect equally children of the rich and children of the poor. The mortality among the poor is no doubt higher than among the well-to-do, which is brought out by comparing the mortality of hospital cases with that of children treated in private practice, ——— This is mainly due to the fact that the child of the poor is seldom seen in the early stage and is often very ill before advice is sought. To make matters worse, in many cases, the condition is aggravated by irregular feeding, and occasionally, by unsuitability of the breast milk.

Heredity : In some cases there appears to be a family tendency towards this abnormal condition. Koplik¹⁵ holds that it may be actually hereditary and, to prove this, reports the case of two sisters whose husbands/

husbands were brothers, both the latter being confirmed dyspeptics of a neurotic type. One of these women had two children (one breast fed, the other artificially), who both suffered from Pyloric spasm and Stenosis. Her sister's baby was artificially fed from the start, but, in spite of this precaution, symptoms of Pyloric Stenosis appeared in this child too, very soon after birth.

It is very probable that in the case of these children a neurotic tendency had been inherited from their fathers, and that the condition of Stenosis was mainly due to a persistent state of Pyloric spasm. It has been noticed by various observers that, occasionally, several children of one family are liable to suffer from gastric disorders in infancy. In some cases this fact may be due to unsuitability of the breast milk — but this explanation does not meet the case of ^{those} ~~these~~ children, who, from the start have been reared on the bottle.

When Peden⁵ in 1889, reported his first case, Cautley tells us, he pointed out that the three previous children in this family had all been "great vomiters". A few years later Henschel described as occurring in one family three similar cases which, Cautley points out, must have been cases of only "partial narrowing of the Pylorus" as they did not present the typical hypertrophic appearance when examined/

examined after death.

Dr John Thomson²⁸ in his most recent paper on this subject, notes that " in one of the cases, the father had suffered as a baby from similar symptoms". In another the mother's brother had died in infancy from what certainly seems to have been Congenital Pyloric Hypertrophy. In four instances there were two members of a family affected.

These instances were drawn from 150 consecutive cases which had been treated by him in Hospital and private practice^{during} the last 25 years.

Judging from the cases that have been treated in the Royal Hospital for Sick Children during the period 1916 - 20 the family history does not usually seem to have any very definite relationship to the occurrence of Pyloric Stenosis; unless, where the Stenosis is caused mainly by a spasmodic contraction of the Pylorus, it has been noticed occasionally that a reurotic tendency has been inherited from one or other of the parents.

Sex : The majority of observers noted that males are more frequently the subject of Congenital Hypertrophic Stenosis of the Pylorus than females. One finds, however, that Rolleston and Hayne²² in 1898, considered that the sexes were equally affected as also did Cautley⁵ in 1906, while quoting his own experience that 5 only out of 16 were females. In both cases they/

they were probably judging from the conclusions arrived at by previous writers, and were not giving us the result of their own observations. Still²⁵ in 1902, judging from his own experience as well as that of others, admitted that the predominance of males is noteworthy; but he did not think that much stress should be laid on this fact.

Choyce⁴ and Hutchison¹³ both found that, in their own experience, males predominated to the extent of 80%. It has been found that in the series of 22 cases occurring in the hospital from 1916 - 1920 the condition was present far more frequently in males than in females, — the percentage of males being 77·3, that of females being only 22·7. If it be proved that males do preponderate, the question naturally arises as to why boys should be more prone to this affection than girls. Until quite recently, no explanation of this problem had been offered. In the September, Lancet of 1919, however, a joint paper by Tyrrell, Gray, and Pirie³⁰ appeared, in which they propounded a new theory, whereby they hold that:- "The male being vagotonic might be expected to show spasm sufficient to cause Hypertrophy more often than the sympathetic^{ic} --- tonic female. " * * * * " Thickening of the Pylorus has been noted in many P.M. examinations of children, both male and female, who showed no symptoms of obstruction during life. The incidence of these symptoms/

symptoms depends upon the relationship, if existing, between the degree of Congenital Hypertrophy and the severity of the added spasm. In girls, the more urgent cause of spasm ——— phimosi ——— does not exist. We are thus led to believe that many children, boys and girls, are born with some degree of Pyloric Hypertrophy, who never develop Pyloric obstruction, and we conclude that the sex difference is more apparent than real. That is to say, apart from the slight excess in the number of males, possibly due to their vago-tonicity, the sex incidence is about equal".

This theory is, at least, an ingenious explanation of why Congenital Hypertrophic Stenosis of the Pylorus has been, and continues to be, so much more frequently diagnosed in boys than in girls. One feels that it must surely be more than mere coincidence as it is a fact which has been noted by so large and varied a number of observers. It will be interesting, in the future, to study the relationship which, according to Tyrrel, Gray, and Pirie, exists between Congenital Pyloric Stenosis and Phimosi, and to note whether circumcision has any beneficial effect on this condition. In two of the later cases of my series, a very marked degree of Phimosi was present, but circumcision was postponed till they had recovered from the Rammstedt operation. Unfortunately, in the case of/

of the others, history does not relate whether or not the boys suffered from Phimosis, so that on this point no statistics are possible.

Age of Onset : The general consensus of opinion is that advice is usually sought when the child is aged from 4 to 8 weeks, and practically never later than the 3rd month. This is probably due to the fact that by the 3rd month the condition will either be settling down, or else the child will be dead. The age of the child when symptoms first occurred is usually found to be 2 - 4 weeks : but in rare cases vomiting does occur right from the first day of birth. In my series of 22 cases the average age when the child was first brought for advice to the hospital was 8 weeks : and the average age at the appearance of the first symptoms was $3\frac{1}{2}$ weeks.

Place in the Family : In 1915 Still¹⁵ said that out of 38 cases, 18 of them were first born children. Hutchison¹³ believes that this condition occurs most often in the first born : and to substantiate this theory he quotes Davidson, who found that this was the case in 10 out of 19 children, making it 53%. Out of 14 of my cases 4 were first born children, i.e. $28\frac{1}{2}\%$: but on the other hand, in some few cases it occurred in the seventh or even the eleventh child, which shows how inconsistent this factor can be.

Nature of feeding : Still, found that 16 cases out of 42 were breast fed. Congenital Hypertrophic Stenosis of the Pylorus is not caused primarily by a faulty diet, as is proved by the fact that most of the infants, when first seen, had been mainly breast fed. Koplik holds that $\frac{2}{3}$ of the children suffering from this disease have been on the breast, at least from the start. This is perhaps natural, as someone observed, when one considers that the majority of babies are tried on the breast for at least the first few weeks of life.

Details are not available in every case, history of my series: but out of 15 no less than 10, for the first two or three weeks after birth, had been given the breast alone. This ratio it will be noticed coincides with the $\frac{2}{3}$ found by Koplik.

In the majority of cases it is found that the child drinks hungrily, and the mother often volunteers the information that the child is always greedy about his feeds. This is probably owing to the fact that the infant really is hungry because practically no food is retained in the stomach. It has already been mentioned that in my series of cases, as in all others, the last child may be affected in a large family of from 7 - 10 members. In such cases it is not uncommon to find that the other children are all healthy and have been reared on the breast without any gastric symptoms/

symptoms whatever. This proves, therefore, that in most cases it is through no fault of the mother's milk that the child begins to vomit because the real cause of the trouble is the faulty condition in the mechanism of the child's stomach: and it matters not what quality of feed or what quantity is given at a time, the symptoms, occur apparently without provocation. A point which is often referred ^{to} by the mother is that the child began to vomit whenever bottle feeding was attempted instead of the breast. It is possible that this may be a factor in precipitating matters ; but, on the other hand, this child with Congenital Hypertrophic Stenosis of the Pylorus *in its life when symptoms might be expected to appear, and the fact that this followed* was probably just about at the stage after the breast was exchanged for artificial feeding, is possibly no more than mere coincidence.

Causation: So varied and so ingenious have been the explanations offered for the causation of the hypertrophic condition of the Pylorus, that one is driven to the conclusion that not one alone of these elaborate theories exactly fits the case. It may be that certain individual cases are accounted for by one special theory, whereas certain others need some entirely different explanation. In a paper on the subject in the year 1899, Still pointed out that four different views had been held of the pathology of this condition:-

- (1) "That the thickening of the Pylorus occurs entirely/

entirely in extra-uterine life, and is the result of spasm from some irritant in the stomach. (Siemon-Dawosky).

(2) That the increase in thickness of the Pylorus is the result of a developmental hyperplasia " a vice of developmental growth " (Adams re Peden's case, Glasgow, Med. J. 1889)

(3) That a primary narrowness of lumen, a Congenital Stenosis, is followed by Compensatory hypertrophy of the stomach, especially the Pylorus.

(Finkelstein, De Bruyn Kops, Meltzer, Landerer and Maier).

(4) That there is " a functional disorder of the nerves of the stomach and Pylorus leading to ill - co-ordinated, and therefore antagonistic action of their muscular arrangements", and that this functional disorder begins ~~in~~ utero. (J. Thomson).

Still, then proceeds to discuss the rationale of these four theories and ^{thinks} the strongest objection to an extra-uterine origin is the improbability, perhaps, impossibility, of so great an hypertrophy, occurring within the short period of extra-uterine life in some of these cases. Then again if the Pylorus can be felt at ^{birth} ~~with~~ or shortly after, and assuming that a Pylorus which can be felt ~~is~~ an enlarged Pylorus, we should have positive proof that the condition was intra-uterine in origin. The second view, /

view, which assumes a primary congenital stenosis as the cause of the hypertrophy, he thinks, hardly needs consideration, because the measurements of the lumen given in several recorded cases of hypertrophy are well within the normal limits, perhaps even above the average. "The Stenosis is the result of muscular contraction during life: in other words it is the result, not the cause of the hypertrophy."

The cases described by Maier and Landerer probably belong to quite a different category: one where there is present a congenital narrowness of the Pyloric lumen, which acts ^{as} ~~as~~ a stricture, produces some general hypertrophy of stomach ~~in which~~ ^{including the} Pylorus: "but in such cases the hypertrophy of the Pylorus is a much less striking feature than in those under consideration". "Developmental hyperplasia the third theory, is not altogether without support, but ——— it is completely negatived if it can be shown, as seems probable, that the Pylorus becomes palpable only some weeks or months after birth: moreover, there is some evidence that the condition is ~~a~~ recoverable one (Finkelstein, Senator & Batten)." Both Ibrahim and Cautley considered that the primary and real cause of the Stenosis was a true Congenital hypertrophy of the muscle in the region involved. ^{Pfandler} "Pfandler ~~or~~ on the other hand, holds that there is no muscular hypertrophy in the wall of the Pyloric Canal, /

Canal, and that the appearances which seem to indicate this, as well as the results which ensue, are all due to a spasmodic contraction of the circular ^{musculature} ~~innec-~~ ~~relative~~ of this portion of the stomach. The Stenosis therefore, according to this author, is not caused by a structural change, but is produced by a functional disturbance of the nervous mechanism which presides over the movements of the stomach."

(Cunningham).³⁵

In discussing the possibility of John Thomson's theory²⁸ of a functional disturbance of the nervous mechanism occurring in utero, Still²⁵ says, that the balance of evidence is strongly in favour of this condition being intra-uterine in origin: and this being so, the choice seems to rest between an unexplained vice of developmental growth, and hypertrophy from increased work. In the third edition of his book "Common disorders of Childhood," published in 1918, he says that he regards it as certain that the obstruction is primarily due to spasm of the Pylorus, and it seems to him most probable that the hypertrophy also is the result of spasm. (The suggestion that a defect of nervous co-ordination leads to antagonistic action, and so to hypertrophy of the muscle of the stomach, is at any rate in accordance with the common experience that elsewhere, hypertrophy of muscle results from increase of work. To this extent also/

also the explanation would agree with that accepted for the adult cases of hypertrophic Pylorus, in which hypertrophy is thought to be due to spasmodic action of the of the Pylorus, the result of some ^{irritation} ~~irritable~~. In the infantile cases, however, there is no evidence of any irritant, and for this reason ^{Dr} Thomson has suggested that the spasmodic action may be due to a functional disturbance. The question therefore, arises, whether there is any evidence to show that functional spasm may occur here, and whether it may occur during intra-uterine life ——— Cases have been recorded in which the *oesophagus* has been found hypertrophied, the thickening ceasing abruptly at the ~~cardiac~~ orifice where there was no evidence whatever of Stenosis (Dr Rollaston's case recorded in the Transactions of the Pathological Society of London) If spasm of the cardiac orifice ~~be~~ the cause of the ~~oesophageal~~ ^{oesophageal} hypertrophy, as Dr Rolleston suggests, then there is ~~in~~ ⁱⁿ such cases ~~a~~ similar ^{condition} ~~cardit~~ to functional spasm of the Pylorus. In 1896 John ^{stated} Thomson his hypothesis that an hypertrophy is due to exaggerated, functioned activity at the Pyloric Sphincter, which has been brought about by a derangement (probably from faulty development) of the nervous mechanism, which regulates the contraction and relaxation of the Pylorus under appropriate stimuli. "If the co-ordination between the stomach wall and the Pyloric Sphincter were disturbed even a little", he/

he continues, "so that the two contracted simultaneously, it is easily seen that both would be continually and greatly overworked. There is also no essential improbability in the hypothesis that co-ordinated muscular actions taking place in utero should occasionally become severely deranged, apart altogether from any previous structural lesion, just as other similar processes are apt to do in later life. It may be asked if there is any analogy to this to be found in the case of other muscular arrangement which act in intran uterine life".

He then describes a Post Mortem examination he had recently made on an infant of 17 days old " who had enormous dilatation of both ureters, very great hypertrophy with dilatation of the bladder, and no discoverable obstruction in the course of the urethra." In an article which appeared in the B. M. J. of September 1902, Dr Thomson elaborated his theory of the origin of the spasm being due to a derangement of cordination and suggested that " it may be a sort of intrauterine "developmental neurosis", i.e. one of those passing disturbances which ^{set} ~~set~~ in at periods when the function interfered with is in process of very active development, being acquired but not yet perfect. " He then compares this stammering of the Pylorus to that which probably occurs ~~in~~ the respiratory system of the child with Congenital Laryngeal Stridor/

Stridor, the young baby who develops "head nodding" in acquiring the art of voluntarily moving his head, and of the stammering gait of a child who is learning to walk.

In his "observations on ^{Congenital Hypertrophy} ~~Congenital Hypertrophy~~ of the Pylorus" written in July 1919, as his contribution to the ^{Oster} ~~Oster~~ Memorial, Dr Thomson refers to his earliest work on this subject written more than 23 years previously. It is gratifying to find that his vast clinical and pathological experience has only served to convince him of his first conclusions as to the Causation of the hypertrophy and his latest words on the subject are here quoted.

He dismisses the theory, held by Hirschsprung, Cautley, and others, that the muscular coat is primarily affected by a 'single congenital redundancy of growth, because he had dealt with this fully in a former paper. It seems to him far more likely that the functional abnormality is to be regarded as the primary element in the process ——— "the muscle being hypertrophied merely because, from an early period of its development, it has been worried into overgrowth by constantly recurring overaction, such as would result from even a slight degree of habitual inco-ordination ———. It is known that the normal foetus swallows a considerable quantity of amniotic fluid during intra-uterine life; and, as this implies

a/

a certain amount of co-ordinated muscular action of the stomach and Pylorus, it is believed that the supposed inco-ordination between these parts begins when the fluid first passes through them. There is reason to believe, however, that the muscular action may not at this period be very vigorous or continuous, and that therefore by the time the child is born only a small degree of hypertrophy will have occurred. After birth, when regular feeding has begun, the force of the muscular action and the inco-ordination will tend to increase so that the hypertrophy will progress much more rapidly than during intra-uterine life. It is in accordance with medical, surgical, and pathological experience that the Pyloric tumour does grow larger and harder while the active symptoms continue. This is just what might be expected, for, as John Hunter pointed out long ago, a tendency to hypertrophy as the result of repeated forcible contractions is "a property of all muscles" and is greater in in-voluntary than in voluntary muscles.

It is extremely probable that tissue - growth of this sort is specially active in early infancy",
 * * * * * "The muscular coat, as we have seen, grows quickly; but the peritoneal tube enlarges comparatively slowly with the general growth of the body, and is incapable of more than a moderate distension. The rapidly thickening muscle, therefore, presses more and more/

more inwards as it grows, and the tube of ~~in~~ mucous membrane is elongated and increasingly narrowed. The stage at which the symptoms become typical in any case probably depends mainly on when the muscular layer has become so thick that, even during relaxation it seriously embarrasses the functional opening of the canal for the passage of food.

Batten, in 1899, after describing his case which made a good recovery with nasal feeding, concluded by saying that he thought the fact that the child recovered makes it probable that Dr Thomson's theory, as to the etiology of the condition, is most likely to be the true one. He had arrived at this conclusion because then the active peristaltic movements of the stomach, which would be started by "deglutition," are relieved by feeding with a nasal tube, then the stomach will tolerate and digest food placed within it. (There is no doubt this was a genuine Pyloric case, because although the child recovered from the gastric condition it died of Bronchopneumonia at 11 months old and ~~that~~ the Post Mortem there was found, in addition to consolidation of the lungs, a typical hypertrophied Pylorus.)

Cautley and Dent: argue that if the condition be due to spasm in all cases, as was held by Pfamdlar, then medical means should suffice to cure the child, and ^{be unnecessary.} Surgical treatment should [^] This, however, is not the case; /

case; and as their view is that the true nature of the condition is probably a primary muscular hypertrophy, which is not the result of spasm, (whereby it is similar to that held by Ibrahim), they hold that "while surgical measures are imperatively demanded in the strongly marked cases, they may also with advantage be adopted even in slight degrees of the affection". After briefly referring to Dr Thomson's theory, of the Inco-ordination of the musculature of the stomach and Pylorus, they point out that the proof that any such repeated forcible contraction of the Pyloric Sphincter takes place, even after birth, in these cases is very far from complete; however, they do not offer any new hypothesis ^{to} ~~and~~ take the place of the one they reject. Cautley, however writing again in 1908, says that he believes there are two different diseases:-

(1) Pyloric Spasm.

(2) True Pyloric Hypertrophy which may or may not be associated with spasm or secondary effects, and may be moderate or ~~excessive~~ ^{excessive} in extent.

He is inclined to think that Pyloric spasm does not cause hypertrophy — and points out that there is no analogous hypertrophy in other parts of the alimentary canal as the result of spasm. He considers that in certain cases the symptoms are due to Spasm of the Pylorus; but where true Pyloric hypertrophy is present the condition is one of Congenital Hyperplasia. "It is possible", he says, "that Nature, in her/

her extreme anxiety to create an efficient Pyloric Sphincter, has over-exerted herself and produced too great an amount of muscular tissue, which is perhaps a reversion to ^{an} ancestral type of stomach. (e.f. gizzard of birds etc.) " He finds that this theory best explains the various types of the disease which come under our notice. Hutchison, however, says, that granted there is present a congenital hyperplasia, "it is difficult to see why obstruction should result, for the mere thickness of the muscular coat, should not prevent its relaxing to allow the escape of the stomach contents when required. Also, infants with Congenital Hypertrophic Stenosis have no special tendency to exhibit malformations elsewhere." He then points out that the supporters of the spasm theory have got to show that spasm is capable of leading to consecutive hypertrophy.

"It would be contrary to pathological law that this should happen, " Hutchison considers, "unless the muscle is not merely contracted, but contracted upon something, so that its fibres are kept in a state of extension at the same time. If it be admitted, however, that the Pyloric canal in infancy is a regulating mechanism which controls the passage of the gastric contents from the stomach to the Duodenum, this difficulty disappears, for in that case it would be/

be comparable to the arterioles which regulate the flow of blood from the arteries to the Capillaries; and it is admitted that prolonged contraction of the arterioles can lead to hypertrophy of their muscular coat.

Haas : according to the New York Medical Journal of May 1919, considers that hypertrophic Pyloric Stenosis is only an advanced degree of Pylorospasm, both being manifestations in the Syndrome of "hypertonia". "The hypertonic infant," he says, "equals a definite clinical entity, characterized by hypertonicity of all the skeletal muscles and by general spasticity of the unstriated muscular system. The hollow viscera may show increased activity of their smooth muscle fibres—this expressing itself in the form of spasm involving practically every part of the digestive tube —, and, depending on the region, may present the symptoms of colic, visible peristalsis, vomiting, constipation, or several of these in combination." This theory, if it were accepted, might perhaps explain the occurrence of a rather unusual case described by Pritchard and Hillier²¹ in the London Medical Press early this year. This was an infant aged 5 weeks with symptoms of Congenital Pyloric Stenosis, in which the autopsy revealed not only a condition of Hypertrophic Stenosis of the Pylorus, but an associated hypertrophy of the cardiac sphincter of the oesophagus and also that of the/

the Ileo-caecal valve. They found no abnormality of the suprarenal glands; but think it possible that there may have been some over - activity of their internal secretion, which resulted perhaps in undue sensitiveness of the neuromuscular mechanism of these various sphincters to stimuli which would otherwise produce only normal responses.

The explanation, offered by ⁸ Haas for this abnormal state of affairs, is that "There has been some disturbance in the physiological action of the vegetative Nervous System, which is made of two parts -- (1) Autonomic (2) Sympathetic, which are normally in a state of balance; but in this condition there is an overaction of the autonomic, resulting in the state of vagatonia". One feels that possibly most of the cases seen by Haas have been those of stenosis produced mainly by spasm, the relief of which would explain the good results obtained by his Atropine treatment. There is perhaps some excuse for this feeling when he himself says that his vagatonic theory does not gain say the occasional existence of true organic Stenosis of the Pylorus. *This much vexed question of the etiology of Congenital Hypertrophy of the Pylorus has been shown in a new and interesting light by the research work*

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of Tyrrell, Gray and Pirie, an account of which recently appeared in the Lancet. They consider that in a typical case of this condition there is undoubted true hypertrophy, which is due to overaction or spasm. The spasm/

spasm inducing the hypertrophy, Pirie suggests " is primarily due to hyper-adrenalism before birth, and that other subsidiary post - natal causes determine the persistence or recurrence of the spasm. This condition is due to a lack of balance between the secretions of the various endocrine organs in the process of their development and involution, which may result either in a relative or an absolute hyper-adrenalism. - - - -

Swale Vincent, Sharpey Schafer, Priestly, Elliott, and many others have shown how important from a developmental point of view are the endocrine organs, and how finely adjusted is the balance between their hormones. If this is so, how much more readily may one accept the possibility of a lack of balance at birth and before, when developmental requirements are urgent and physiological processes so active. If this disturbance in balance results in a relative or absolute excess of the suprarenal medullary hormone, spasm of any non-striated muscle may result. Keith has shown that both the Pylorus and the medulla of the suprarenal gland become differentiated, at about the third month of intrauterine life. Since excessive suprarenal secretion produces spasm in non-striated muscles this would allow plenty of time for spasm, induced by any excessive suprarenal secretion, to bring about hypertrophy. He also states that before birth the gland is larger than the kidney, and at birth may be the/

the same size. After that it rapidly reaches its normal relative size. —————. A gland which undergoes. Such marked changes in its development and involution may easily have had its balance upset a long time before birth, with resulting Hyper-adrenalism. This may exist in widely varying degrees and would determine the amount of hypertrophy present at birth.

It cannot be claimed that in all cases the hyper-secretion is controlled by changes in the gland itself. The ready response of its secretion to stimuli from the semilunar ganglion would lead one to expect that in some cases there is hyperadrenalism following excessive stimulation of the splanchnics. Tyrell, Gray and Parsons have shown the effect of excessive stimuli passing to the Sympathetics. Their work gives a possible explanation of the added Pyloric spasm in the frequently associated condition of Phimosi^s". Pirie, concludes his hyperthesis by saying:- "There is clinical evidence to show that there is pancreatic insufficiency in cases of congenital Stenosis. Add to this the evidence of Sharpey Schafer that the suprarenal gland is antagonistic to the pancreas, and it would seem that hyper-adrenalism, relative or absolute, would inhibit pancreatic secretion. There is then good reason for justifying the assumption of pancreatic insufficiency, which we know is a powerful factor in producing pyloric spasm. Given hyper-adrenalism, there is at once sufficient cause for the primary/

primary hypertrophy induced by spasm and for the perpetuation or reinduction of this spasm by pancreatic insufficiency".

In their summary of conclusions drawn both from this article and from the one that follows, (written by Tyrrell, Gray, and Pirie conjointly), they say that the final results in the closure of the Pyloric orifice are:-

- (1) Absence of acid chyme in the 1st part of the duodenum, leading to (2)
- (2) Failure of secretin formation, leading to (3)
- (3) Suppression of pancreatic secretion. These factors further induce to (4)

(4) Inhibition of the normal Pyloric relaxation and establishment of the "vicious circle" - (4)→(1)

Pirie ends his discussion by raising the question whether ~~if~~ hyper-adrenalism may exist, why not hypo-adrenalism? and he then suggests that the etiology of the whole question of Intestinal Stasis, may have to be reconsidered from this point of view. In conclusion he remarks very wisely:- "This fact is clear, we must not study children as young adults. Their physiology is entirely different, and their symptoms should not be measured by similar symptoms in adults. Many of the problems they exhibit must be studied from the developmental point of view, and in this connexion none of their organs are more important, or so easily upset by faulty overaction during the course of their development and involution, than the Internal secreting glands."

S Y M P T O M S.

Vomiting. The characteristic features of this symptom are not described by the earliest writers on the subject, and we know little more than that vomiting was the chief symptom complained of. Peden⁵ however in 1889 noted that vomiting began three days after birth, and was persistent and characteristic---in that large drinks were immediately rejected whereas small ones were collected and vomited later on. Dr John Thomson,²⁸ in describing his second case in 1896, went one step further, and noted that the child usually vomited half an hour after a drink, but sometimes took several feeds without vomiting and then, after an interval, vomited all the collected feeds at once. A third case came under his care in the following year, and of this child he records that severe vomiting set in, which occurred in a projectile manner through both mouth and nose.

The vomiting was frequent and occurred immediately if ^{the} feed consisted of more than 2 oz.

After being fed by a stomach tube with peptonised milk for a day or two there was an improvement in the symptoms and the vomiting seemed to have been checked; but Dr Thomson made this important discovery, that when the tube was introduced at the end of a two hours/

hours interval, instead of being empty the stomach contained nearly 2 oz. of sour turbid fluid with small soft curds. This proved, therefore that although the stomach was able partly to digest the milk there was practically no absorption going on, and no passage of food through the Pylorus.

Another interesting point which was noted was that, although the child did not vomit if fed by a stomach tube, this symptom immediately returned whenever an attempt was made to return to bottle feeding. In his article on "Congenital Gastric spasm" which appeared in 1897, Dr Thomson noted that the rapidity and ~~certainly~~ of vomiting seems to depend on the amount, not on the nature of the feed; and he also drew attention to the "retentive" nature of the vomiting. Cautley⁵, writing two years later, stated that although the vomiting usually depends on the amount, and not on the nature of the feeds, still, there are apparently some fluids which seem to be more readily retained than others. Having mentioned several ~~ly~~ easily digested fluids such as Peptonised milk, as being the least likely to result in vomiting, he then proceeds to quote the advisability of thin farinaceous fluids as used by Gran of Christiania. This point is of particular interest just now when Sauer and others are advocating the use of thick cereal feeds made with Farina, which, they argue, cannot be vomited/

vomited so easily as milk feeds owing to its immobility; and, therefore, since it remains in the stomach it can be moved along by the slow persistatlic contractions of the gastric wall. One can understand such a method being ~~was~~ successful where the obstruction is due mainly to a spasmodic contraction of the Pylorus, just in the same way as the Duodenal Catheter treatment of Hess might overcome such an obstruction; but it seems difficult to believe that such treatment would open up the stenosis which must be present in a true case of Congenital Hypertrophy of the Pylorus.

In a typical case then the child is born apparently normal and healthy, and remains so during the first week or two of life. During this time there are often no symptoms at all; but occasionally there may be slight regurgitant vomiting from birth, which fact ~~is~~, rather apt to mask the typical symptoms and thus confuse the issue. In perhaps the majority of cases the child is breast fed and there is probably no irregularity in the method or times of feeding to ^{account} ~~amount~~ for the symptoms. About the third week, sometimes earlier sometimes later, vomiting sets in. ~~It~~ ^{at} first occurs at long intervals but these gradually become shorter and shorter until ^{the infant} ~~it~~ vomits after nearly every feed. When the stomach, however, has appreciably dilated as a result of the obstruction, the vomiting somewhat changes its character and becomes of the "retentive" type; so that it occurs only at long intervals, sometimes/

sometimes only once or twice in the 24 hours, and consists often of more than one feed at a time. The mother sometimes volunteers the information that the feeds are "lashed up"; and on further enquiry it is perhaps discovered that so forcible is the ejection that the vomited material "shoots" through both mouth and nostrils, and may reach over the side of the cot a distance from 2 - 3 feet. This is what is usually termed "projectile" vomiting. It may be that a change is then made in the diet, and for a day or two the symptoms disappear and apparently all goes well. The improvement however, is only temporary and after a few days a relapse occurs and the symptoms return perhaps with redoubled energy. This point is important because it differentiates a true hypertrophic Pyloric Stenosis case from one when the vomiting is due merely to a condition of ~~the~~ Dyspepsia, in which the symptoms would slowly but surely disappear under such simple methods as regulation of the diet, and perhaps occasional lavage of the stomach. The infrequency of the vomiting has led, in some cases to its being almost overlooked, --- and as the wasting in some cases is often very marked it is not difficult to understand how sometimes the real condition passes unrecognised and the case is diagnosed as one of obstinate marasmus.

Still points out that the "retentive" nature of the vomiting may be due to the stomach dilatation; but/

but he considers that ^{it} ~~he~~ means more than this, and that it points to variation in the degree of obstruction, which seems most naturally explained by varying degrees of spasm of the Pylorus at different times.

According to Rotch²⁴ and Murphy, the reason why the vomiting ~~is~~ of a projectile nature is that, in a typical case, it is not simply due to the running over of a well-filled stomach. On the contrary, it is expulsive in character, for the reservoir is contracted forcibly in its effort to drive food along its normal channel.

The infant ~~seems~~ sometimes to be in pain, which is relieved by emptying the stomach, and in such cases there is apparently no accompanying nausea as the child resumes its feed as soon as it has vomited.

The vomit consists usually of a large amount of fluid containing soft curds. In cases where the stomach is much dilated the fluid is said to be frothy, but I have not personally seen this. When there is present, (as a result of the obstruction usually), any gastric catarrh, this is usually indicated by mucus being present in the vomit. Should any bile be present, this should be looked on as conclusive evidence, that there can be present no real obstruction of the Pylorus. The time that vomiting occurs appears to be not consistent with the time of the feed; and this inconsistency appears not only in cases of Pyloric/

Pyloric Stenosis as a whole, but also in the individual feeds of every case. In other words, it is noticed again and again that at one time the child may take its bottle greedily, and so fast that it is hardly finished before the vomiting begins. On another occasion, the same child may suck in a leisurely fashion, so that the feed becomes retained longer, perhaps half an hour, or even till after the next feed --- in which case it will be found that the vomit is much larger than the amount taken with the last bottle.

Several observers have noted that in certain cases the child may vomit after a feed, but if another feed be given immediately after this has occurred, the second feed is retained without any difficulty. The reason for this phenomenon is probably that after the exaggerated spasm of the Pyloric Sphincter, which must occur during the act of violent projectile vomiting, there is a corresponding reaction exhibited by relaxation of the Pyloric spasm sufficient to overcome temporarily, the stenosis. This, however, could only occur in a case of Partial obstruction, such as might be present if there were a moderate degree of hyperplasia of the Pylorus and added to this a condition of spasm of the circular muscle fibres.

According to most authorities it is typical of this condition that the tongue remains clean and the breath/

breath sweet in spite of the vomiting; but where dilation of the stomach has occurred and this has been followed by gastric catarrh, it is only to be expected that the breath becomes ^{sour}~~sew~~ and the tongue is usually coated thinly with a white fur.

CONSTIPATION.

Hutchison,¹³ in his Schostein Lecture delivered in October 1910, when describing this particular symptom called it "Pseudo-constipation", because it is not due to any intestinal disorder, but simply to the failure of food to find its way into the bowels at all. Still²⁵ in 1918 holds that:- Persistent vomiting accompanied by chronic constipation is almost pathognomonic ~~gnomise~~ of a true case of Congenital Hypertrophic Stenosis of the Pylorus. It should be easily distinguished from the vomiting which is due to a faulty diet, because in such a case there exists in addition, not constipation, but frequent loose green motions. The character of the motions is not mentioned in some of the records of the very earliest writers on this subject: but of his first case Dr John Thomson notes that there was constipation with the occasional passage of dark motions containing mucus, and there was in addition some uneasiness on passing water.

At the Post Mortem examination of this child it was found that the Duodenum and intestines were normal but empty, ~~th~~^{is} accounting for the constipation. The symptom of **Dysuria**, which he mentions as being present in his second case too, is probably due to the anuria, a natural sequence to the fact that practically no/

no water passes the stenosed Pylorus, and little or none is absorbed from the stomach. Anuria seems to be a fairly constant symptom and has been noted by most of the subsequent observers.

In his third case the motions are described as being small, green and slimy, which tallies with the description of Rotch and ^{Ladd} ~~Food~~ ⁶ in 1905 in which ~~they~~ ^{they} say the motions consist of bile stained mucus containing little or no faecal material; and with a similar one by Koplik in 1908 and Crozier Griffith in 1905.

Dr Thomson found that in his third case a temporary improvement resulted from tube-feeding, so that much more fluid was absorbed; this being proved by the fact that a previously scanty urine was now passed more freely.

Cautley ⁵ in 1899 stated that in his first two cases the bowels had been ~~very~~ costive, but he considered that constipation was not invariably present. This view he enlarged upon in 1906, noting that the state of the bowels is variable, but that by far the commonest condition is to have persistent and increasing constipation. "Sometimes" he says, "the stools are brown or dark green, sometimes tarry. Occasionally multiple watery stools are passed, this being the result of enteric catarrh set up by irritation from retained stomach contents, which have now been enabled to pass the Pylorus. Miller and Wilcox ¹⁸ have said/

said that this diarrhoea is due to the fact that the intestines have become atrophied and functionless through disuse, so that no absorption occurs, and the stomach contents thus pass though practically unaltered. This may be the explanation of any transient diarrhoea that may occur; but it is ^{difficult} ~~different~~ to believe in any other way, in face of the fact that in the cases that make a good recovery there is apparent then no lack of Intestinal function.

In Peden's case there was well marked constipation and also in Finkelstein's, but in those of Hirschsprung, Gran, and Scharyzer, the constipation was at any rate preceded by diarrhoea, and occasionally the bowels were never anything but loose. It is possible that in such a case there might be a super-added acute enteritis which would cause the diarrhoea and would probably account for the death of the child in some cases. It is obvious that in such an one the obstruction could be neither complete nor permanent; and its degree may have been determined by the varying amount of additional spasm present in the Pyloric musculature.

Meltzer in 1898 described a case which he had diagnosed and had operated on. The child was fairly constipated but Meltzer apparently did not realise the true nature of the constipation which he treated by giving doses of Calomel. It is surprising to find that/

that the bowels in this case responded to such treatment seeing that the constipation was due, not to Intestinal Stasis but to the fact that stomach contents were not passing the Pylorus. With the improvement in the bowel condition ~~he~~^{she}, too, noted that the urine becomes ~~less~~ scanty in amount.

It was stated by Herman in 1907 that in a marked case the bowels never move spontaneously and the motions are small in quantity, thick, pasty, and dark coloured. "The amount of faecal matter", he says, "gives a very good idea of the degree of Pyloric Stenosis. In mild cases the bowels may move regularly but the motions are only in small amount."

Batten writes of his case which made such a good recovery after careful feeding with a nasal tube, that as the vomiting stopped the constipation improved, and for the first time the child began to pass partly digested motions. A difficult case is described by Hunter in which there was neither constipation nor diarrhoea, but one small green motion was passed daily.

The motions in a case described by Townsend²⁹ in 1908 were evidently black, stringy, and meconium-like, even on the twentieth day after birth, but there was never any faecal matter present. A gastro-enterostomy was performed and on the second day after the operation a little faecal matter was passed.

In/

In an article on Hyper-adrenalism, which appeared in the Lancet of September 1919, Pirie writes that:-
 "The small hard dry stool so characteristic in these cases, is due as much to the lack of secretions as to the small quantity of food passing through the Pylorus.

Thus, there is clinical evidence to show that there is pan-creatic insufficiency in cases of Congenital Stenosis. Secretory inhibition explains the hard consistence of the stool. The size of the stool, in a measure, indicates the degree of obstruction at the outlet of the Pylorus; while the consistence indicates the degree of secretory inhibition which is present.

No Cathartic should ever be given as the cause of constipation, is the lack of bulk as well as lack of secretion due to obstruction of ^{the} Pylorus ——— a small oil enema or rectal lavage is all that is necessary. After relief from obstruction, either by medical or surgical means, even though fed on peptonised milk, the child had frequent, loose, greasy, stools for several days (Still, has pointed out the grave danger of diarrhoea following such an operation). This, however, is not due to any inflammatory change in the mucus-membrane but to the presence of incompletely digested fats. (Still, says that these babies take fats badly). This faulty digestion/

digestion is due to the fact that the function of the Pancreas has been inhibited for a long time and it is not possible for this to be resumed immediately the obstruction is removed. On the contrary it takes a correspondingly long time before the Pancreas Secretes sufficient ferments to digest the proteids and fats."

EMACIATION.

Dating from the time of Williamson onwards, the majority of observers have commented on the progressive emaciation which occurs in children that were healthy at birth, and ^{who} sometimes for the first week or two had no symptoms.

Dr John Thomson¹⁸ in 1897, in reviewing the cases of Congenital Pyloric Stenosis that had already been recorded, remarked that they had usually been full time infants, well grown and well nourished. Thus it was proved that this condition, although congenital, does not seem to interfere with the nutrition of the foetus.

Cautley⁵ described a case of his which occurred in 1899, and in which the child tho' "healthy born" began to vomit at three weeks and died a week later in a state of extreme emaciation. In his description of the typical symptoms of such a case he says of emaciation that ——— "the temperature is sub-normal and the skin cold and clammy. The child lies in a condition of whining lethargy with half open eyes, and takes no interest in its surroundings. Food is often refused, and occasionally it cries from pain and rouses itself sufficiently to vomit."

In the same year, Batten in recording his now famous/

famous case stated that the child had been vomiting since the age of five weeks, the abdomen was flaccid, the fontanelle depressed and the temperature sub-normal.

Harper described a case in 1905, and he considered that in this disease marasmus is usually progressive ——— "the child rapidly wastes, the skin becomes yellowish brown and the tissues dry and shriveled". This is interesting when compared with the description by Murphy and Morse in 1908, of a child which was small and thin, but had a fair colour; and with that by Townsend in the same year where he said that although no food apparently passed through into the intestine, still the child showed but little loss of flesh or colour (Diagnosis of Congenital Hypertrophic Pyloric Stenosis was confirmed at the operation).

Apropos of this feature of the emaciation ¹³
Hutchison wrote in 1910;— "The child rapidly loses weight and soon becomes extremely emaciated. The wasting however, is not like that met with in Cachexia. The child is not anaemic but simply looks starved, and often remains surprisingly bright in spite of it. In this respect it differs from the wasting of ~~the~~ most forms of organic disease, and to the experienced eye, the difference often suggests a correct diagnosis". The reason for this characteristic must be that, in spite of the fact that practically,

practically no digestive processes are going on, still just sufficient is absorbed to keep the child alive; and the colour remains healthy unless there be present any superadded condition of gastric catarrh or enteritis.

Herman¹² wrote in 1907, that the infant is usually apathetic and very much emaciated and continued thus:- "The weight progressively diminishes in marked cases (3 or 4lb in a few weeks). As the inanition becomes more pronounced the amount of food that can be given without producing alimentary intoxication becomes progressively smaller. An effort should, therefore, be made to ascertain what is the minimum amount of food necessary to prevent a loss in weight".

In the following year, Koplik stated that emaciation and progressive loss of weight are constant signs in this disease and that generally the appearance of the child is typical with its sunken eyes, hollow cheeks, and retracted abdomen.

The possibility of mistaking a true case of Pyloric Stenosis for one of marasmus from faulty feeding was emphasized by Still in 1915. He thinks that until recently these cases of Pyloric obstruction have mostly passed for the common marasmus of infancy, or have been attributed to unsuitable feeding. The history, he concluded, may differ but little, if at all, from that of many a common place case of infantile/

infantile wasting:-

The general appearance of the child in long standing cases, according to Pirie, is characteristic of a case of marasmus^s, whatever the cause of wasting.

PHYSICAL SIGNS.

The Two signs which are usually found in a well marked case of Pyloric Stenosis are:-

(1) Visible Gastric Peristalsis.

(2) A Palpable Pyloric tumour.

The importance of these signs can hardly be over-estimated, because if they are really definite there can be no doubt but that the infant exhibiting them is suffering from a hypertrophied condition of the Pylorus. On the other hand, the absence of either or of both these signs is not by any means proof that Pyloric Stenosis does not exist in this particular case. The stomach may be too greatly dilated to show up waves of Peristalsis, or in an earlier case the Abdominal wall may be still too thick for the waves to be visible even though they do occur. Similarly, the Pyloric tumour may not be easily palpable through a thick wall; or it may be tucked away up under the liver, where the fingers cannot reach, and thus, although present, it may be impossible to feel it.

(1) Visible Gastric Peristalsis.

As one would expect this Sign was recognised much earlier in the history of Congenital Pyloric Stenosis, than was the 2nd one, which is sometimes very difficult to elicit, and in some cases cannot be recognised at all, even by a skilled diagnostician. It is not surprising that the very earliest descriptions of this disease contain no reference to either of these Signs, and probably Finkelstein in 1896 was the first to record the fact that one of his cases had exhibited both these Signs, and that the Peristalsis of the stomach was very distinctly visible. Two years later Meltzer¹⁶ gave a fairly lengthy description of this sign and stated that:- "After nursing, the peristaltic waves could distinctly be seen passing over the Epigastrium from left to right. The bulging upper part of the abdomen contrasted sharply in appearance with the sunken lower part. At the commencement of the 4th week the stomach was unusually contracted when empty, and showed peristalsis when full. Gradually a state of atony of the stomach developed - it became more and more permanently/



permanently dilated extending over the larger part of the abdomen, and no more peristalsis was seen. At this point vomiting seemed to diminish but with no sign of food passing into the intestine or being absorbed from the stomach". It is in the stage of "attempted compensation" that, according to Meltzer, peristalsis occurs; and after the stomach has given up trying to overcome the Pyloric obstruction and has subsided into the stage of "atony and permanent gastro-ectasy" - then it is that the formerly visible waves of Peristalsis gradually cease to appear.

Batten's² case in 1899 showed very distinct peristalsis, and marked ~~dilatation~~^{dilatation} of the stomach, which fact is rather at ~~variance~~^{variance} from what has just been quoted of the observations made by Meltzer. But in Congenital Pyloric Stenosis more than in any other condition one has to judge each case individually, and it is quite impossible to make sweeping assertions ~~except~~^{expect} these to embrace every single case that may occur.

In the following year Nicoll described his case, which became almost classical owing to the fact that it was the first genuine case of Pyloric Stenosis to recover as a result of operation. When Nicoll first saw this infant at the age of 5 weeks, emaciation had occurred to such an extent that through the attenuated Parietes there stood out the form of a considerably dilated stomach with occasional peristalsis. Before this/

this, when the body was still well nourished it was impossible to detect these ~~operacterieties~~ ^{Characteristics}. Nicoll at this time observed the following facts:-

" (1) Peristaltic gastric waves. (2) Periods of normal dilatation of the stomach (after a meal?), alternating with periods (after vomiting?), during which the organ may be felt like a ball, firmly contracted (spastic contraction?). (3) Marked abnormal dilatation of the stomach, associated with, and rendered the more prominent by, a collapsed condition of the rest of the abdomen, consequent upon the empty state of the intestine".

Cautley and Dent⁷ in 1903 described the appearance of typical peristaltic waves in the following words:- "Peristaltic waves started at the stomach, passed onwards to the *Pylo*rus and paused there ———, then continued onwards down the *Duodenum*". And again three years later, Cautley observed that visible Peristalsis may only be present after feeding. If, therefore, any difficulty should be experienced in seeing definite waves, the child should be given a fair sized feed, and then flicking the stomach lightly with one finger or applying a cold object, may stimulate the sluggish contractions, and thus produce the sign that is looked for. "If the stomach wall is much dilated", he continues, "and the walls weakened, the wave does not cause so marked/

marked a bulging of the abdominal wall. Peristalsis may be seen in other varieties of Pyloric obstruction, and in wasted infants with dilated stomachs; but in these it is much less characteristic".

This fact was also noted by Still in 1905 and by Herman in 1907.

Still says of Peristalsis that it is most likely to be seen immediately after food, and that the abdomen should be watched during a feed as waves may become readily appreciable as soon as food enters the stomach. It may be necessary, however, to knead or stroke the epigastrium repeatedly before any peristalsis becomes visible. In continuing, he says, that "The slow wave like movements are quite different from the irregular, voluntary contraction of muscles of the abdominal wall, which are frequent enough in a squirming infant. Peristalsis, in the gross form here described, if occurring during the first few months of life, is peculiar to Congenital Hypertrophic Stenosis of the Pylorus, — , it can be seen sometimes at a distance of from 2 - 3 yards, and may even be remarked on by the nurse or the mother. A fuller description is given in the 1918 edition of "Common Disorders and Diseases of Childhood" which is well worth quoting:- "A rounded lump, varying in size from half a large walnut to half a Tangerine orange, rises at the left costal margin and passes very slowly across the Epigastrium, slightly downwards towards the right Hypochondrium. Before this lump has yet/

yet reached the mid-line, a second similar lump is already at the left costal margin; and sometimes before these two have yet disappeared in the right Hypochondrium, a third is already appearing on the left side, ——— so that at one time three bulging eminences are seen, like a chain of hills, extending across the Epigastrium. They move so slowly that at times they may be seen to pause altogether, but each in turn fades away in the right Hypochondrium, and a succession of these bulgings may continue to appear for 1 or 2 minutes. A very feeble peristaltic wave may occur, just after feeding in infants with chronic vomiting and constipation, but it is very different, (as Herman says), from the distinct tonic contractions which are present at Pyloric Stenosis, and to be seen at all it is necessary to examine the abdomen obliquely in a good light".

6 .

In Crozer Griffiths' case in 1905 the peristaltic waves were always particularly active when the child was approaching an attack of vomiting. In spite of gradual improvement under careful dieting and gastric lavage, vomiting returned at intervals, the stomach remained dilated, and peristalsis was often active.

In the same year, a case was described by Harper in which peristalsis was excited by the introduction of anything into the stomach, or even by exposing the abdomen to the air. The wave could be felt moving across/

across the abdomen if the hand was laid on the stomach. On rare occasions he saw a "Retrogressive" wave, which evidently started from the Pylorus and travelled towards the Cardiac end of the stomach. This peculiar wave was often accompanied by vomiting. One wonders if perhaps this retrogressive wave does not perhaps quite frequently occur in this type of case, being well obscured, however, by the grosser contraction of the abdominal muscles, which must occur at the same time to facilitate the act of vomiting.

This "reverse" peristalsis, which is said to occur just before the act of vomiting, was referred to by Koplik in 1908, who remarked that Ibrahim had never seen it, and that he himself had never been able to make it out, and that if it did occur at all it must be instantaneous. "In some cases the peristalsis was so extreme that, just previous to vomiting, the stomach would erect itself on the abdomen and divide itself distinctly from what appears to be the Pyloric end of the stomach. It would then contract and vomiting would take place." Koplik disapproves of the custom of trying to elicit peristalsis by palpating the stomach and causing the "platch" noise, and he regards these methods as fruitless in considering the symptomatology. Herman considers that gastric peristalsis, when present is pathognomic of congenital Hypertrophic Stenosis of the Pylorus. "At the beginning of the disease", he says/

says, " the abdominal tissue conceals the movements of the parts beneath, but after the child has become emaciated peristalsis is invariably present. Visible peristalsis may persist after vomiting has ceased, proving that a partial obstruction at the Pylorus, alone, is not sufficient to cause persistent vomiting."

29

The record of Townsend's case in the same year is interesting because he notes that there was present no visible Peristalsis, and no ~~Pyloric~~ tumour could be felt, although a post mortem examination confirmed his diagnosis that the child was suffering from a well marked ^{hypertrophic} Pyloric Stenosis.

The condition described by Magid in the following year was apparently not the usual type of Peristalsis because, in his words:- "Occasionally the epigastric area suddenly bulged out as if it had become distended with gas. This condition lasted about a minute, during which the child screamed as if in pain, and on one occasion vomited some fluid just after". This sounds rather similar to Koplik's description, in which it seemed occasionally as if the stomach erected itself on the abdomen, contracted, and then vomiting ensued.

Hutchison, lecturing on this subject in 1910, stated that he looked on gastric peristalsis as the most characteristic sign of all. "This vigorous peristalsis," he considers, "is pathognomonic in the child, as in the adult, of some obstruction at the/

the outlet of the stomach, and is, therefore, of great diagnostic value. Curiously enough it does not seem to cause much, if any, pain, and the child will often lie quite placidly, whilst the waves course in succession across the organ." This, it will be noticed, is a very different account to that given just previously by Magid, where the child screamed as if in pain, and one's own feeling is that very probably in the latter case there was largely an element of spasm, causing the Stenosis, and that it was the Spasmodic contractions of Pylorus which gave rise to a considerable degree of pain. Hutchison's description, on the other hand, is probably an accurate picture of the condition, as seen by the majority of observers, in a genuine hypertrophic case.

(2) Palpable Pyloric Tumour.

This is looked on by many as the all-important sign, and this alone is the one in the presence of which a definite diagnosis may be formed. Probably this is a very extreme view to take up, but it is quite true that a person skilled in the art of palpating the Pylorus, might detect the presence of a tumour in a case where extreme dilatation of the stomach might obviate the appearance of visible Peristalsis. He would thus recognise a case of Pyloric/

Pyloric Stenosis which otherwise might conceivably have been missed. It is perhaps a mistake to ^{lay}~~lay~~ a great deal of stress on one of these phenomenon and not on the other. When present, both these signs are characteristic of this condition, and ~~if~~ sought for consistently, and at the most suitable times, then it should be difficult to go very far wrong in the diagnosis.

28

Dr. John Thomson, in his third case, on careful ^{palpation}~~palpitation~~, found a small, hard, very movable tumour in the Epigastrium. He pointed out that Finkelstein, in the previous year, had made out clearly by ^{palpation}~~palpitation~~, the hard hypertrophied Pylorus, as an easily movable tumour lying in the epigastric region, somewhat to the right of the mesial line. Finkelstein apparently proved its anatomical connections by the passage of a catheter, and by the injection of air into the stomach. He had felt the tumour 2 cm. above and somewhat to the right of the umbilicus.

2

Batten said of his case that, on deep ^{palpation}~~palpitation~~ in the Right Hypochondrium about a finger's breadth outside the nipple line, he could feel a firm transverse mass. in shape like the Pylorus. This could not always be palpated unless at the same time peristalsis was in progress. As the child improved the Pylorus was less distinctly felt than on admission, and sometimes not at all. This, I gather, was due to gradually increasing/

increasing thickness of the abdominal wall, and not to any disappearance of the Pyloric tumour, which seems to have been a typically hypertrophied one when seen at the autopsy (~~death~~ having occurred from Bromhopneumonia at 11 months after the child had apparently made a complete recovery, as far as symptoms ~~from~~ from the Pyloric condition). This same fact was observed by Harper in 1905.

Nicoll was able to palpate a tumour in his case in 1900 and this was eventually confirmed at the time of the operation. He remarked that in the recorded cases this tumour has usually passed unrecognised, and that it is probably present only in those few cases in which the hypertrophy of the Pyloric tissues has been marked, and the abdominal parietes very thin.

Cautley and Dent⁷ in 1903- in describing the peristalsis, noted that the waves passed onwards from Stomach to Pylorus and paused there before passing on towards the Duodenum, "Deep down under the liver, at the point of temporary pause, is a rounded ~~incorable~~ ^{moveable} tumour". They believe, however, that a Pyloric tumour, even when of considerable size, may not be palpable. In 1906, Cautley⁵ described the hypertrophied Pylorus as a hard lump about the size and shape of a filbert, or rounded like a small marble. It is situated, he finds, $\frac{1}{2}$ inch/

inch to ^{the} right and $\frac{3}{4}$ inch above the ^{umbilicus} ~~unbrilius~~, half way between the ^{umbilicus} ~~unbrilius~~ and the Costal margin. Occasionally it is lower down and may, therefore, be mistaken for an enlarged gland. Sometimes it is difficult to reach, because it lies buried beneath a large liver, and in a well nourished child it may be very difficult to palpate. The greater the wasting, therefore, the more palpable the Pylorus, and the smaller the chance of successful operative treatment. A palpable tumour, he considers, is almost absolute proof that it is a genuine case of Congenital Hypertrophic Stenosis of the Pylorus that is being dealt with.

Still²⁵ wrote an interesting description of the Pyloric tumour in 1905, ——— "on deep palpation", he said, "usually just outside the right nipple line and about a third of the distance from the umbilical level to the Costal margin, a ~~hard~~ lump, barrel-shaped, and seeming perhaps about $\frac{3}{4}$ inch long, by $\frac{1}{2}$ inch wide, is to be felt at intervals. This tumour, the hypertrophied Pylorus, behaves exactly like an Intussusception in its variations of palpability. It is during peristalsis that it is to be felt; at other times the muscle is so soft that no amount of tactus eruditus will enable you to feel it. For this reason palpation should be made during the visible peristalsis of the stomach. Occasionally, even when/

when there is no visible peristalsis in progress at the time, the Pylorus can be felt evidently undergoing contractions, being palpable and hard one moment and completely lost the next. Such contraction may be excited by gently kneading with the tips of the fingers pressed deeply into the abdomen in the situation of the Pylorus."

Bloxland³ in 1905, described the tumour as a hard elongated lump, about half the size of the terminal phalanx of the little finger, felt mid-way between the umbilicus and the right Costal margin. It was not always palpable, could often be felt to form under the finger, and was most distinct when gastric peristalsis was most obvious.

In 1907 Herman¹² found that in his case a little to the right of the midline, midway between the ensiform Cartilage and the navel, a small tumour about the size and shape of an olive could be felt.—— In its normal position, he believes, the Pylorus is not palpable, but the dilatation of the stomach, which follows obstruction, together with possibly a laxity of the suspensory ligaments, makes palpation possible.

Rotch and Murphy, in the same year, said that, if present, a Pyloric tumour is diagnostic of the condition; but that inability to feel it is not conclusive negative evidence as to its existence; because it may be obscured by a large liver, or by a rigid/

rigid abdominal wall, or possibly it is only felt when in contraction. The size of the tumour, he remarks, need be no indication of the degree of obstruction present.

In the following year, Koplik noted that during a period of contraction and peristalsis a small hard tumour may be felt, cartilage like in consistence, situated sometimes beneath the liver or its border, and running directly down towards the umbilicus. Situated deeply against the vertebral column is the Pyloric end or valve of the stomach, as it meets the Duodenum.

The method of palpation, as described by Hutchison in 1910, is that one should try to get underneath the stomach and compress the Pylorus against the right side of the vertebral column. Often, he says, it is best felt by trying to pinch up the deeper structures. Sometimes the Pylorus is so tucked up under the liver that it cannot be felt, but, if palpable, feels like a hard lump, almost the size of a hazelnut, but somewhat elongated.

A palpable Pyloric tumour is looked upon by Tyrrell Gray, and Pirrie³⁰, as the one certain sign, and they consider that failure to find it leaves the diagnosis in grave doubt. The most usual site for it, they find, is just outside the outer border of the Rectus in the Trans-Pyloric plane. Deep palpation is necessary/

necessary as it often lies well back, tucked in beside the vertebral column. Occasionally it is higher up, in which case care must be taken to exclude an enlarged Quadrate lobe of the liver. A valuable clue to its situation is the right limit of the peristaltic waves. It feels like a marble, which rolls away from the examining finger as it is touched. If the stomach is much dilated it may overlap the Pylorus, and thus render it ~~very~~ difficult to palpate, particularly if the tumour be a small one.

They give a valuable hint for palpation in a ~~different~~ ^{difficult} case where there is, say, a large liver or a deeply placed Pylorus. Their plan is to turn the baby on its face and to allow the slightly flexed body to rest on the palm of the hand, thus partly relaxing the abdominal wall. The viscera, therefore, fall forwards and the fingers can be passed behind the liver, and it is thus possible to explore the whole region. A practical point that they mention is that it is always best to examine with the left hand, from the left side of the patient.

DIAGNOSIS.

Little remains to be said of the actual diagnosis of Congenital Pyloric Stenosis in infants, now that the typical symptoms and signs have been described.

The greater diagnostic value of one characteristic of this disease over another has perhaps been a little over-rated; particularly that of the presence or absence of a Pyloric tumour. The fallacy of basing one's diagnosis solely on this point is borne out in certain cases where the presence of an hypertrophied pylorus can be discerned by the few only who possess great skill and experience in this difficult form of palpation.

Under such circumstances, the unskilled will probably fail to detect the presence of a tumour. If this, then, is to be the basis of their diagnosis, such a case must be inevitably missed, and the child thus lose its one chance of recovery. On the other hand, if a broader view were taken, whereby all the symptoms would be embraced and given their due share of importance, there would then be less risk of a genuine case passing unrecognized. A definite Syndrome should be looked for. If it be discovered that a child, apparently "healthy born", started after/

after about the 2nd week of life, to vomit persistently, that the vomiting tends to be projectile and is gradually becoming of the "retentive" type; that it is accompanied by marked and increasing constipation and loss of weight, and that there is present a very marked degree of emaciation, then the doctor's suspicions should be aroused.

The history of projectile vomiting should, if possible, be verified by personal observations, and the stomach tube should be used two hours after a test feed to form a rough idea of the degree of the obstruction, if any, that is present. If, in addition to these facts, typical Gastric Peristalsis be observed on more than one occasion, then the diagnosis should be fairly correctly formed within a few days, and the most suitable treatment decided upon accordingly. Should a Pyloric tumour be palpated then there will be the satisfaction of almost absolute certainty. I do not think, however, that a negative finding should ever be looked upon as conclusive evidence that the case in question is not one of Congenital Hypertrophic Stenosis of the Pylorus, - unless the diagnostician has implicit confidence in his own skill of interpreting accurately what he feels on palpation.

Cantley and Dent, in 1903, bore out this view to/

to a certain extent by saying that a pyloric tumour, even when of considerable size, may not be palpable. They subsequently mentioned certain complications which may be present, and to a small extent perhaps alter the clinical picture:-

1. "Some gastritis may (but only rarely) supervene.
2. Dilatation of ^{the} Stomach may occur.
3. If any toxæmia dependent on constipation occurs, the temperature may be irregular."

Still, in 1905, emphasized the importance of an early diagnosis to the chances of successful treatment, and then mentioned a few fallacies which may occur in the diagnosis of visible peristalsis:-

"(a) Voluntary, irregular contraction of muscles of the abdominal wall when the infant is squirming, as often happens when the abdomen is exposed for observation.

(b) Peristalsis of the Transverse Colon, if slightly transposed."

Still apparently does not agree with those who describe a feeble peristalsis in marasmic infants, (which, however, has no real resemblance to the typical, gross peristalsis of Pyloric Stenosis), because he says that Peristalsis probably does not occur in infants apart from Congenital Hypertrophic Stenosis of the Pylorus.

DIFFERENTIAL DIAGNOSIS.

Pyloric Spasm is perhaps the most likely condition to be confused with Congenital Pyloric Stenosis, because the ^{symptoms}~~systems~~ may be very similar; and indeed, in rare instances it may be almost impossible at first to distinguish them. Usually one is guided by comparing the degree of severity of the symptoms, and whether Peristalsis, if it be present, is really typical or not. The vomiting in a case of pure spasm may be projectile but it is seldom of the "retentive" type. The diagnosis would be assisted by passing a stomach tube 2 hours after giving a test feed, because although the residue might be considerably more than normal, it would likewise be considerably less than in a case where organic obstruction is present at the outlet of the stomach. If an enlarged Pylorus can be definitely palpated then there need be no further doubt, and it should thereby be possible to establish a correct diagnosis of Stenosis due to a congenitally hypertrophied Pylorus. There are other cases, however, where it is by no means so easy to be sure - the pylorus, for instance, may perhaps be vaguely but not quite definitely palpable. In such a case, provided that the child is not visibly getting/

getting worse nor having any serious loss of weight, the only justifiable course is to treat the case tentatively by careful dieting and gastric lavage until some definite sign of Hypertrophic Pyloric Stenosis should make its appearance. Cattley⁵ tells us in 1906 that there is a greater danger of Pyloric Spasm being diagnosed as hypertrophy than vice versa. It is necessary, he says, in forming a diagnosis of Congenital Pyloric Stenosis, to exclude all such common disorders of infancy as:- Simple Regurgitation, Gastric Catarrh, Vomiting and Constipation from a faulty diet, and habit vomiting. He looks upon the presence of a Pyloric tumour as almost absolute proof, but points out that although the diagnosis is quite easy in many cases, in others it is almost impossible. A comparison given by him between a case of Pyloric Spasm and one of Congenital Pyloric Stenosis forms a very helpful little table for diagnostic purposes:-

(See page 10)

PYLORIC SPASM.

PYLORIC HYPERTROPHY.

Vomiting (rare for 2 or more feeds to be retained)

Vomiting, projectile(2 or 3 feeds retained, then vomited)

Peristalsis - slight or absent

Peristalsis - well marked.

Stomach - rarely dilated.

Stomach - often dilated.

Constipation - unusual, may be diarrhoea.

Constipation - very marked.

Pyloric Tumour - none.

Tumour - palpable = hypertrophied Pylorus.

In addition to these differences, Koplik¹⁵ added, in 1908, that in a case of Pyloric Spasm there may be one or two stools daily, which usually contain a milk residue. In a true case of hypertrophy however the occasional stool consists of bile-stained mucus containing little or no faecal matter. In this condition too he found that the Pylorus is usually distinctly felt as a large nodule: whereas in Pyloric spasm the nodule, if palpable at all, is very much smaller in size.

Miller and Willcox¹⁹ hold that as an immediate diagnosis is sometimes a matter of great difficulty, the case may need prolonged watching before any definite conclusion can be arrived at, and that in such a case, therefore, a gastric analysis may be a valuable aid to the diagnosis. They published an article in 1908 in which they stated, that, as the result of a series of investigations, they had found that differences exist in the gastric secretion in various cases of infantile wasting. "These differences", they continue, "may be shown to correspond to 3 clinical groups:- (1) Pure Marasmus (which we may term "atrophic dyspepsia"); (2) Hypertrophic pyloric stenosis; (3) Cases of pyloric spasm unaccompanied by hypertrophy which we find to be cases of acid dyspepsia". The investigations have been made on the stomach contents withdrawn half an hour after a test meal had been given.

Their results they have tabulated in the following manner:-

Class.	Quantity.	Mucin.	Ferment Acidity.	Total Acidity.
(1) Atrophic Dyspepsia (Marasmus)	Small	Absent	Low	Low
(2) Pyloric Stenosis	Large	Present	Usually high	Variable (usually low)
(3) Acid Dyspepsia (Pyloric Spasm)	Large	Absent	Low	High

The stool, in a case of Marasmus, they found to be usually loose and green. In Pyloric spasm there was a tendency to constipation, which, however, was much more marked in a case of hypertrophic stenosis where the stools are usually small, dark coloured and marble-like.

Rolleston and Crofton-Atkins³⁴ were convinced in 1900 that in order to be sure of the diagnosis the pyloric tumour must be palpated, because, in its absence, it is impossible to exclude all other ~~cases~~^{causes} of vomiting. Coutts,³¹ on the other hand, wrote ten years later that a palpable pylorus is less often made out than visible peristalsis or projectile vomiting. He then remarked that "the value of this sign is largely discounted if it be true, as some assert, that mere spasm of the Pylorus can lead to a palpable swelling, differing in no wise from one due to a definite organic hypertrophy of the circular muscular coat of the Pylorus". Other observers have pointed out that when a tumour is palpated, if it be due to pyloric spasm, the tumour may be felt to contract under the fingers, or even completely to disappear. The tumour of Congenital hypertrophic stenosis of the pylorus, however, during the act of palpation is felt to remain unaltered both in consistence and in/

in position.

Dr John Thomson²⁸ in his most recent article on this subject, pointed out that his worst cases of Pyloric spasm have been in girls. He has noticed too that a child suffering from this spasmodic obstruction cries often as if in great pain, which rarely happens in a child suffering from a hypertrophic condition of the Pylorus.

I had almost omitted to mention a very rare condition, but one which might make the diagnosis more complicated -- that of Duodenal Stenosis. In such a case, however, there would be bile present in the vomit, a fact which would at once rule out the possibility of the symptoms being due to Stenosis of the Pylorus.

I have been fortunate enough to see a case of Duodenal Stenosis, which has impressed on me the difference between the two conditions. The case in question was that of a male child aged 4 weeks with a history of vomiting bile stained fluid ever since birth.

He died 5 days after admission. The post mortem examination revealed the fact that the Duodenum had been twisted on itself at the junction of the 1st and 2nd parts, and had become matted down in this position by adhesions. The Caecum was tucked away up on the left side underneath the stomach. No other abnormalities were found.

P R O G N O S I S.

As to the prognosis of this condition it is quite impossible to generalize, and one can only form an opinion of the prospects of recovery or otherwise in each case individually.

In 1899 Cautley⁵ looked upon the prognosis as extremely bad if the treatment be purely medical - ~~even should the observation~~ ^{obstruction} be incomplete. Under such conditions, he believes that the average duration of life, dating from the time of onset of typical symptoms such as projectile vomiting, is about 4 months. He finds that the shortest period of this kind recorded was 17 days, and that the longest was 6 months; but, he adds, the cause of death here was Tuberculosis.

Operative treatment, on the other hand, Cautley believes may have excellent results, if undertaken before the child has become marasmic.

Koplik in 1902¹⁵ observed that "recovery depends on the resistance of the patient, the skill with which the case is managed, and the power of the sufferer to overcome the baneful effects of inanition. He has come to look upon recovery by medical means as being not incompatible with a marked degree of congenital hypertrophy and stenosis, just as others have considered this impossible."

Koplik is perhaps a little too optimistic, and yet one feels that Cautley is somewhat of an extremist in the opposite direction.

MORBID ANATOMY.

Dr John Thomson²⁸ in his article "On Congenital Gastric Spasm" in 1896 briefly ~~received~~ ^{reviewed} the historical cases which had already been recorded. In the Morbid Anatomy Section of his paper he said that:- "There is always more or less emaciation due to the extent to which the incessant vomiting has interfered with nutrition * * * * The alimentary canal below the Pylorus is perfectly normal. The oesophagus is sometimes noticeably dilated, sometimes of normal calibre. In none of the cases was any other congenital abnormality noted. The stomach is generally considerably enlarged; and while its walls at the Cardiac end are as thin as, or thinner than usual, over the rest of the organ they are much thicker than normal. The Pylorus itself is much enlarged, and looks and feels like an almost solid fusiform, or even oval mass of muscular tissue. When looked at from the Duodenum the Pyloric opening seems almost closed, the mucous membrane being puckered by the contraction of the hypertrophied muscular wall: and, although it readily allows the passage of a probe, even firm pressure on the stomach is not sufficient to drive fluids through it into the bowel. In none of my cases, certainly, was there any fibrous stricture present/

present at all. The whole narrowing of the passage seemed to be due to compression by the hypertrophied muscle. The mucous membrane seemed indeed rather smaller in extent than in a normal Pylorus, but the constant compression to which it had been subjected would easily account for this. In each case it was voluminous compared to the size of the lumen which it lined. In all the cases which were examined microscopically (except Finkelstein's), the thickening of the Pylorus was due mainly to great hypertrophy of the circular layer of muscular fibres. Finkelstein's case formed a remarkable exception to what seems to be the rule in this matter, as in it hypertrophy of the longitudinal muscular layer constituted the main cause of the thickening. The serous coat is unchanged. The ^{submucous} ~~subcutaneous~~ coat is sometimes greatly thickened, sometimes slightly so, and sometimes it is normal. The mucous membrane is either quite normal, or it shows merely such changes as would be expected to follow the constant violent vomiting and the attempts to feed the child in a variety of ways."

Meltzer¹⁶ in the year 1898 found in his case that had had an unsuccessful Gastro-enterostomy performed, that the thickening of the Pylorus was largely due to the presence of dense fibrous tissue in/

in the submucosa and to a hyperplasia of the inner muscular layer. The undue prominence of the gastroduodenal valve, as seen from the duodenal aspect, is due, he says, to the fibrous hyperplasia in the submucosa.

Still in 1899²⁵ discussed the post-mortem appearances in three cases which he had examined at Great Ormond Street Hospital. The microscopic appearances of the Pylorus he found very similar in all three cases. The increase in thickness he found to be almost, if not entirely, limited to the muscular coat, the increase being most marked in the circular layer. The thickness of the longitudinal layer of muscle fibres in two cases was actually within the normal limits, but in the third case distinctly exceeded the normal thickness. He found no definite increase of fibrous tissue, but the bundles of muscle fibres in the circular layer were perhaps rather more distinctly marked off by the fibrous trabeculae than is common in the normal Pylorus. The submucosa in the first two cases was normal in thickness; but in the 3rd case, the one which had an hypertrophied layer of longitudinal muscle fibres, had also a submucosa of twice the normal thickness. He did not think that there was any "Condensation" of the fibrous tissue here. The/

The mucosa and the serous layer were apparently normal in all three cases, and in the mucosa there was no evidence of inflammatory infiltration past or present.

Still found that even the normal Pylorus shows a variation in thickness, which occurs mainly in the circular fibres. There is very little variation however in the thickness of the submucosa normally. He believes that the average diameter of a normal Pyloric lumen is 3.5 - 4 mm. In referring to the funnel-like shape of the Pylorus on the stomach side, and of its likeness to the lower end of the cervix uteri on the duodenal side, a fact which has been greatly emphasized by certain writers, he agrees that such is the case but that it occurs also in the normal stomach, though perhaps to a lesser degree.

Cantley in 1899⁵ stated that the Pylorus is represented by a very definite tumour which varies a little in size, but may be taken roughly as about the size of the last joint of the little finger, and about an inch long. On external examination with the fingers, he finds that the limits of the tumour can be definitely fixed both on the duodenal and the gastric side: to the touch it ends more abruptly on the duodenal side, but there is also a very distinct limit to it on the gastric side.

The/

The occlusion of the lumen seemed almost complete in some cases and was due to the contraction of the hypertrophied muscle assisted by the thickening of the mucous membrane, which is thrown into one or more longitudinal ridge-like folds extending the whole length of the thickening. Cantley had at first thought that there was an hypertrophy of the mucous membrane, judging from naked eye appearances, but he found microscopically that the thickening is only apparent and is due to the contraction of the walls of the tube. He found a very marked hypertrophy of the circular muscle fibres, a moderate increase in the amount of intermuscular connective tissue, and a slight increase in thickness of the external longitudinal layer of muscle: He questions Finkelstein's statement that the thickening in his case was due to hypertrophy of the longitudinal muscle fibres and thinks that probably the appearance was due to the section having been cut obliquely.

Hutchison¹³ observed in 1910, that the thickening of the wall of the Pyloric canal is due almost entirely to an enormous development of circular muscular fibres, although the longitudinal layer is also thickened, and the mucous membrane and submucous coats to some extent. The muscle fibres, he says, are not only increased in number, but each individual fibre is also broader than normal.

T R E A T M E N T.

The baffling nature of Congenital Hypertrophic Stenosis of the Pylorus is mainly accountable for the extraordinary variety of methods that have been devised for treating this disease, the number of them alone plainly indicating how disappointing, usually, have been the results. Some few people still have the mistaken idea that there are two distinct lines of treatment - that offered by the physician, and that by the surgeon. They imagine, therefore, that should the child be under the care of a physician the treatment would be conservative; whereas if he were in other hands the treatment would be surgical. No doubt there was a time when that was the case more or less; but now by the word "treatment" we understand a far bigger thing than either the one or the other, - we understand a combination of the two, which requires very skilful co-operation on the part of both surgeon and physician. The child suffering from this disease, in all probability is brought for advice first to the physician. Once the diagnosis of Congenital Hypertrophic Stenosis of the Pylorus has been definitely established he would be wise to consult with a Surgeon, so that between them they might decide whether in this particular case an operation/

operation was or was not indicated. Should an operation be decided upon the interest of the physician in this child has by no means ended. His experience in the highly specialized art of managing a difficult "feeding" case will all be required for the post-operative ~~the~~ stage of treatment - that time when, even in the most favourable cases, for days and sometimes weeks the infant hovers between life and death. From the foregoing it might be thought that I look upon an operation of some kind as a necessary part of the treatment in every genuine case of Pyloric Stenosis caused by hypertrophy. That it is essential in the majority of cases is perhaps true; but there is a small minority in which the results of operation are far from encouraging, and it is in these few cases that one sometimes regrets not having persevered longer with more conservative measures. Unfortunately, in our present state of knowledge, it is almost impossible to discriminate, or to decide with any certainty which cases are likely to do well surgically, and which are likely to be failures. Moreover, it sometimes happens that the very picked ones, which one hoped would make ~~the~~ successful operation cases, have eventually proved disappointing; whereas apparently hopeless cases have recovered.

It/

It is about hospital cases alone that the treatment still remains such a vexed question. About a case seen in private there need be no hesitation because the results of surgical treatment of this disease, under favourable home conditions, are becoming more and more encouraging - and far outweigh the successes recorded by non-operative means. It appears somewhat of a paradox that this should be so, when one thinks of the many advantages prevailing in a hospital with its irreproachable surgical technique, and its experienced nursing staff trained to attend to every want of the infant both night and day. And yet this is a well known fact and has been recognized by almost every observer, that the mortality in hospital, particularly of operation cases, greatly exceeds that occurring in private practice.

One of the chief reasons for this, I imagine, is that the hospital type of case is usually the child of poor, ignorant parents, who do not realize the seriousness of the condition until severe symptoms and marked emaciation have already occurred. When advice is at last sought the child may be practically moribund with scarcely any possible chance of recovery. It is only logical to expect that in better circumstances the parents are more enlightened, and seek advice as soon as anything out of the ordinary, /

ordinary, such as persistent vomiting, has been noticed. There is thus every chance of radical treatment being given while the disease is yet in a fairly early stage, with a proportionately greater chance of recovery.

We have been told by many writers that the great danger after operation to hospital cases is the risk they run of developing an acute enteritis, which, should it occur in their weakened state, is naturally very apt to prove fatal.

There is probably a great deal to be said for this theory, because the post-operative treatment, in this hospital at least, is frequently conducted in a medical ward in which there are possibly several babies suffering from varying degrees of enteritis. No matter how elaborate the precautions it does happen now and again that a diarrhoea infection runs round the ward, and as might be expected, it is the puny little infant struggling to recover from the effects of an operation, who is most likely to fall a victim to this infection and develop a fatal diarrhoea. For this reason I think it might be a wise plan if the child were kept in a surgical ward until the abdominal wound should be healed. The reason for this proposal is that in a surgical ward the average patient is/

is fairly healthy apart from the surgical condition present, and there are usually fewer tiny babies than in a medical ward, thus lessening the risk of a diarrhoea infection occurring.

Doubtless there are difficulties in the way, but none serious enough to prove real obstacles. The feeding might be a difficulty because, in a hospital such as this, the nurses in the surgical wards are generally used to older children and have perhaps not yet had the exact experience that they would require to manage successfully a difficult case such as this must inevitably be. A solution to this problem would be arrived at if it were possible always to have a special nurse put in charge of the post-operative treatment of a Congenital Pyloric Stenosis baby, until the danger period should be past. The surgeon would be wise to arrange with the physician, who first saw the case, that he should superintend the "feeding" and, if possible, continue to interest himself in the case after the child has been discharged ^{from hospital.} In all cases where there is a suitable mother and where the home conditions are at all favourable the child should be discharged as soon as possible after the wound has satisfactorily healed. This procedure is practically what does occur in this hospital, with this one difference that the physician at the present time usually takes the case back/

back into his own ward, and supervises the post-operative treatment there. It would be an interesting experiment to see whether a slightly different routine might at all influence the mortality rate, which, according to our hospital statistics, is still alarmingly high.

The progress of treatment for this condition, starting from its earliest beginnings, forms a very interesting chapter in the history of Medicine. The pioneer cases were not usually recognized in time for any definite treatment to be attempted, because, to begin with, there was no known method of diagnosis and it was usually only at the post-mortem examination that the true nature of the condition was revealed.

Schwyzer in 1896¹⁶ advised that as soon as a diagnosis of this disease had been made the treatment should be surgical, the operation being either that of Loreta or else a Gastro-enterostomy (in his case by means of a Murphy's Button). Although this operation was not successful, the very fact of its failure taught a valuable lesson, and convinced Meltzer of the fact that the use of Murphy's button is unsuitable in infants, that even an ordinary Gastro-enterostomy might be disappointing in its results, and that the operation of choice is Bilioth's Pylorotomy.

In/

In the following year Batten² described a case he had cured by means of "nasal feeding". This was, so far as is known, the first child to have recovered under conservative treatment, and indeed it is probably the first case of recovery on record.

In 1900 Nicoll²⁰ operated on Dr Ritchie's case. He performed a modified *Loreta*, and later on remarked in his notes that this was "probably the first successful case of operation for this affection". In the same year Rolleston and Crofton Atkins³³ were of opinion that, judging by the encouraging results of Batten's treatment, nasal feeding should be tried in a slight case, but a really definite case should be operated on.

Cantley and Dent in 1903⁷ advocated the operation of Pyloroplasty rather than that of *Loreta* because it is a more definite operation and the amount of injury done to the parts is known, while it can be done every bit as quickly. A Gastro-enterostomy they find less suitable than Pyloroplasty because it takes much longer and it necessitates a considerable exposure of the abdominal contents.

Still, in 1905,²⁵ advocated medical treatment, and again in 1918 he said that he thinks many cases can be cured medically, but there certainly are some where an operation is needed to save life.

Three years later Koplik¹⁵ stated that he prefers conservative treatment, and feels that his chances with nature are as good, if not in some cases better, than with the operative measure. He has not found much benefit from gastric lavage, and in certain cases, thinks that this routine treatment may actually lead to an exacerbation of symptoms. Harper⁹ a few years before this, had pointed out the value of giving small feeds frequently, which contrasted with Meltzer's advice in 1898¹⁶ that the amount at each feed should slightly overstep the normal capacity. In 1907, Sutherland³⁴ stated that he had not found the slightest benefit from medicinal treatment; although he had pushed Opium and other such drugs to the point of their full physiological effects, it had been without the slightest effect on the stenosis. He found rectal and subcutaneous salines of great value in cases where the tissues were parched and marasmic.

Hutchison was converted in 1910, he says¹³ to uphold the value of conservative measures. He says he fully believes that there may be some cases which would have been saved by operation that die without it, but knows of no means by which such cases can be recognized. "As a practical policy, therefore, one is obliged either to operate upon all cases/

cases in which the symptoms and signs are well marked, or upon none of them." He is convinced that the former plan would result in a much heavier death-rate than the latter.

Hess in 1912¹⁰ described a method of dilating the lumen of the Pylorus by passing a Duodenal Catheter, and in suitable cases was actually able to feed the infant by passing the liquid directly thus into the duodenum. It has been argued, however, that such a procedure might be beneficial where the stenosis is only slight, but success can hardly be expected in a severe case where the Pyloric lumen will barely admit even a silver probe.

In 1918 Sauer²⁶ described a method of gradually loosening the constriction of the Pylorus by means of thick cereal feeding. He found that this thick immobile ~~Farina~~ Farina cannot be ejected from the stomach in the ordinary manner that a milk feed would be vomited, and therefore remains in the stomach long enough to be gradually moved further on by the slow peristaltic contractions of the gastric wall. It is interesting to find that in 1896 Gran⁵ had noticed that in his second case the digestion of milk was more imperfect than that of farinaceous fluids.

Haas in the following year⁸ considered that hypertrophic/

hypertrophic pyloric stenosis is only an advanced degree of Pylorospasm, both being manifestations in the syndrome of hypertonia. For this reason, he tells us, treatment by Atropine is followed by rapid subsidence of symptoms, the results being so prompt and regular as to constitute a specific action. One would expect good results from this treatment in a case of simple Stenosis due mainly to spasm, but it is difficult to understand how Atropine could have any effect on a stenosis due to an enormously hypertrophied circular layer of muscle.

In September 1919, Tyrrell Gray and Pirie³⁰ described their usual routine in treating a case of Pyloric Stenosis. Extreme cases where severe symptoms have been present from birth, and frail, puny babies should be operated on at once. In all other cases palliative treatment should first be adopted. Under this subheading are included various measures:-

- (1) Peptonized milk feeds, (2) Gastric Lavage,
- (3) Circumcision if indicated, (4) Subcutaneous saline and glucose, (5) Chloral Hydrate to allay spasm.

In the majority of cases this palliative treatment may be continued with safety for 10 - 12 days, but in a very weakly emaciated child it should be stopped in 48 hours if no relief of symptoms has occurred in this /

this time. This period of 10 - 12 days, they continue, coincides with the re-establishment of the pancreatic function in successful cases. Soon after the obstruction is relieved the stools become more milky, greasy and more frequent, owing, we are told, to the fact that emulsification and saponification cannot proceed in the absence of pancreatic secretion. It would be interesting to know if this looseness of the bowels corresponds to the tendency to diarrhoea which has been recorded as having occurred in most cases at a certain stage after operation.

Tyrrell Gray and Pirie consider that failure to improve after 10 - 12 days palliative treatment, in boys is an indication for operation, because all causes of spasm have already been removed, and the only obstruction left is that caused by the hypertrophy itself. In girls the onset of the condition is slower and they find that improvement is correspondingly slow, and that treatment must therefore be more gradual than in the case of boys. They are quite convinced that a Rammstedt operation is the most beneficial surgical treatment for a case of hypertrophic ^{pyloric} ~~phloric~~ stenosis. They point out that Rammstedt of Münster had operated successfully in this manner and his name was associated with this operation in 1913. I have since discovered an article³² in the/

the Deutsche Medizinische Wochenschrift - Berlin - the October number of 1912, in which he describes having performed an operation of this kind in 1912. The operation consists of making a longitudinal incision throughout the whole length of the pyloric tumour and extending inwards through serous and muscular coats, stopping short only when the mucous coat has been reached. When the mucous membrane is seen to bulge outwards into the walls of the incision it will be easily understood how great may be the relief obtained by this simple procedure. Care must be taken to carry the length of the incision well beyond the hypertrophied portion of the Pylorus on the stomach side. At its other end however it need not be carried so far, and in fact it might be safer to stop just short of the termination of the hypertrophied part, because of the risk of dividing the thin walled duodenum by encroaching too far. This should be quite sufficient to relieve the obstruction especially as it is the severing of the hypertrophied part adjoining the stomach which is the most vital part of the operation. The pylorus is then returned to the abdomen without suturing the incision.

The mucous membrane is so thick that there is no fear of leakage occurring into the peritoneal cavity, and in this manner the relief obtained from the previous/

previous obstruction should be almost ideal. The great advantages of this operation are its simplicity, the speed with which it can be done, the small amount of handling and consequently low degree of shock.

Tyrrell Gray and Pirie tell us that their experience, clinically and pathologically, shows that after a Rammstedt operation the Pyloric hypertrophy rapidly disappears.

POST-OPERATIVE TREATMENT.

In certain cases the infant becomes somewhat collapsed as a result of the shock of the operation. Stimulants and salines will probably be required in such a case to tide over matters temporarily.

Cattley in 1906⁵ gave a good scheme of treatment which he found very successful:- "Immediately after the operation a nutrient enema containing 1 oz. each of peptonized milk and water with 20 drops of brandy if needed. These should be repeated every 4 hours for 2 days, every 6 hours for 2 days, and twice a day for 2 days - after which they may be omitted.

A teaspoonful of hot water should be given by the mouth every $\frac{1}{4}$ hour for 6 - 12 hours, and after that a similar quantity of whey. Subsequently the diet is slowly increased to 2 teaspoonfuls every $\frac{1}{4}$ hour, a tablespoonful every $\frac{1}{2}$ hour, 1 oz. every hour, and finally 2 oz. every 2 hours. After that it may be strengthened by the addition of cream, or gradually replaced by peptonized milk, and milk and water, until the child is on an ordinary diet. Great care must be taken not to overfeed these children in ^{the} early stages of convalescence on account of the liability to enteritis, especially in the marasmic cases." It should be noted that Cattley was describing/

describing the after treatment as he would arrange it to follow such an operation as Gastro-enterostomy. It might be considered rather unnecessarily elaborate for the after-treatment of a Rammstedt case, where there has been no injury to or opening of the gastric mucosa. In support of this it may be interesting to note the after-treatment prescribed by Tyrrell Gray and Pirie³⁰ to follow a Rammstedt operation. During the 10 - 12 days, the time which the pancreas usually takes to regain its function, they give only peptonized milk:- 1 oz. 4 hours after the operation, and a similar amount every 4 hours for the first 24 hours. During the second 24 hours an ounce and a half is given every 3 hours. After this an amount is given according to the age and weight of the child. They have never found it necessary to give smaller feeds.

When the stools cease to show incompletely changed fats (usually about the 10th - 12th day) they change the feed to citrated modified milk.

The change in the stools, they believe, indicates a return to the normal outflow of pancreatic secretion. "Beyond this point they are fed as normal babies."

NEW SERIES OF CASES.

The first Rammstedt operation to be performed in this hospital was done by Mr Mitchell on the 31st August 1916. Since then this operation has been repeated in 9 other cases, all of the latter being performed by Mr Fraser. With his permission and that of the Honorary Physicians of this hospital, I have made out a series of 22 cases, which includes all cases of Congenital Hypertrophic Pyloric Stenosis that have been treated here during the years 1916 - 1920 (inclusive). Unfortunately my statistics are very incomplete, as some of the notes had been taken very briefly and did not refer to certain points which one would like to have enquired into. The following table has been arranged by me in an attempt to give concisely the salient features of these 22 cases.

TABLE./

The Cases that gave occurred in R.H.S.C. from 1916 - 1920.

No. of Case	Sex	Date when first seen	Age in weeks	Age in weeks at On-set of Vomiting	Constipation	Peristalsis.	Pyloric Tumour	Order of Child in the Family	Medical Treatment	Ramstedt. Age at time of operation.	Surgeon	Recovery or Death.	P.M.	Age in weeks at Death	Age at Discharge	Breast fed or bottle	Remarks.
1	F	4.2.16	16	(Projectile) +	+	+	-	?	+			R			20½	?	
2	M	31.8.16	5	3	Motions hard & dark	+	-	1		6	Mr Mitchell	D		6		Breast for 3 weeks	
3.	F	11.8.16	6	+	+	-			+			D		20			
4.	M	9.2.17	8	+	+	+			+			D	+	8½			
5.	M	21.3.17	10	+	+				+			D		11			
6.	M	27.2.18	12	7	+	+		2		13½	Mr Fraser	D		14		Breast	1 stillbirth & 1 miscarriage.
7.	M	2.3.18	8	3	+	+	+	3	+	+		D		13			
8.	M	18.4.18	12	+	+	+	+		+			D		12½			
9.	M	6.8.18	8						+			D		10½			
10.	M	3.9.18	2	pro- jectile	Small, hard & thro nose black & mouth			6		7	Mr Fraser	D		7		Malted milk.	2 stillbirths & 1 miscarriage.
11	M	23.4.19	8	+	+	+	+		+			D		10			
12	F	18.5.19	12	+	+	+			+			D		15			
13	M	1.7.19	8	4	+	+		7		9	Mr Fraser	R			26	Breast	R.I.H. & well marked Phimosi.

No. of Case	Sex	Date when first seen	Age in weeks	Age in weeks at Onset of Vomiting	Constipation.	Peristalsis.	Pyloric Tumour	Order of Child in the Family	Medical Treatment	Ramstedt. Age at time of operation.	Surgeon	Recovery or Death.	P.M.	Age in weeks at Death	Age at Discharge	Breast fed or bottle	Remarks.
14	M	22.7.19	10	4	+	+	+	2		11	Mr Fraser	D		13		Breast	
15	M	5.11.19	7	2	+	+		1				D	+	8		Breast	
16	M	21.11.19	7	projectile through nostrils	+	+		1		8	Mr Fraser	D	+	11		Breast	Phimosis
17	M	30.12.19	4	Since birth	+	+	+	1		5	Mr Fraser	R			8		Very tight Prepuce.
18	M	6.4.20	6	2	Diarrhoea	+	+	11		11	Fraser	D	+	11		Cow's milk & Glaxo	
19	M	26.4.20	5	3	+	+		Second illegitimate.		Too ill for operation		D	+	6			Temp. rose to 105.8 before death.
20	M	14.5.20	5	+	+	+		5		10	Mr Fraser.	R			13		Improved best after sent home.
21	F	7.9.20	10	+	+	+		2	+			D	+	11½			
22	F	29.7.20	12	+	+	+	+	2		12	Mr Fraser.	D	+	14			

There are one or two points that have been made out from this series which may prove of interest. It will first be noticed that males predominate to the extent of 17 out of 22, which works out at 73% approximately.

The average age when first seen at this hospital was a little over 8 weeks, and it has been found that the average age when symptoms were first noticed, was $3\frac{1}{2}$ weeks. The nature of the feeding has not been described in every case, but of the 14 where it has been referred to one finds that for at least the first 3 weeks, usually for longer, the child had been breast fed (78.5% breast fed.) Projectile vomiting, Constipation and visible gastric Peristalsis occurred practically universally, except in case 18 where, in spite of a typical condition of hypertrophic stenosis, diarrhoea was complained of rather than constipation. In only 7 out of the 22 cases has it been noted that the Pyloric tumour could be quite convincingly palpated; but in spite of this fact in practically every case the diagnosis was verified either at the operation or by a Post Mortem examination. It is not possible to say what proportion of the boys suffered from Phimosi because no reference has been made to this condition in the notes. In the only 3 cases in which I have examined ^{especially} ~~particularly~~ for this condition, I have found a particularly well marked /

marked Phimosiis, accompanied in one of them by a Right Inguinal Hernia.

The total mortality in this series is somewhat disturbing. Out of 22 cases there have been only 4 recoveries, which implies a total mortality of 81.8%. One unoperated case is reported to have recovered in 1916, and 3 operation cases have recovered during the past year. This may be mere coincidence, or it may be that the nursing staff are beginning to understand better how to manage the after-treatment as they gain more experience in it. It may, however I think, be looked on as a distinctly encouraging sign that the results from the latest cases are improving, and one hopes that before long the death rate may gradually but surely be lowered.

The mortality for the cases that have been operated on, (Rammstedt), stands at 70%; that of the unoperated ones being 91%. It is only fair, however, to point out that 4 of the cases treated medically were so far gone that no treatment, either medical or surgical, could possibly have saved them, and they died within 4 - 9 days of admission. No comparisons, therefore, can really be drawn, because the surgical cases were, in a way, picked ones; and in certain instances they had previously had perhaps a week of careful dieting and treatment by gastric lavage, all of/

of which would tend to make more auspicious the chances of recovery. If any comparison is to be drawn it would be fairer to subtract 3 deaths from the 11 which occurred under Medical treatment, as in these 3 cases the infants were almost moribund on admission. This would bring the mortality down to $66\frac{2}{3}\%$.

In Dr John Thomson's last series consisting of 100 consecutive cases, occurring both in hospital and in private practice, he found that of his hospital cases the total mortality where no operation had been performed was 74.2%. In those where an operation (usually a gastro-enterostomy) had been done, the mortality was 75%. One can definitely say, therefore, that the surgical statistics for this disease have improved since the introduction of the Rammstedt method. There is no reason why we should not look forward to an even better state of things in the future.

I have Mr Fraser's permission to mention this interesting fact that his experience of employing the Rammstedt operation in private is that it has been attended by 100% recoveries. As all the Rammstedts in this series, with one exception, have been performed by him, this proves conclusively the fact that the chance of recovery is infinitely greater when/

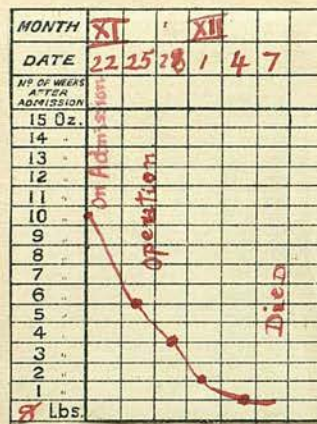
when the case can be attended to at home rather than in hospital.

Number 22 is an instructive case from this point of view that if ever a child had a good chance of recovery from hypertrophic stenosis this one did - and yet she died within 2 weeks of the operation. In this case there was no delay that could be blamed for the failure. A Rammstedt operation was performed almost immediately the diagnosis had been made, which was only a day or two after the infant was first seen. The general condition was good, and the operation appeared to be completely successful, but the symptoms were unrelieved and in a fortnight the child had died. I would draw attention to the fact that it is not always delay in performing the operation which is accountable for unsuccessful results, because this particular operation was performed under comparatively ideal circumstances - and yet the unexpected occurred.

During the past year an unusually large number of these cases seem to have been treated in this hospital, and as I have had the good fortune to see most of these children, I propose now to give a slightly more detailed account of some of the cases in which I have been personally interested.

I have taken from the table on the preceding pages the/

Weight Chart of
Case 16.



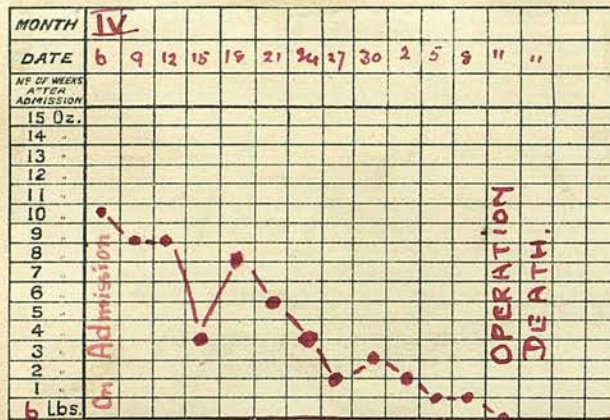
the six most recent cases which had Rammstedt operations performed. In 3 of these cases a complete recovery occurred: in the other 3 however the results were more disappointing and death eventually ended the struggle.

The cases that did not recover will first be briefly described.

CASE 16. (see table). *See Plates 3 & 4 (page 118a)*

Male, aged 7 weeks. Family history good and patient is the only child. Complaint is that of "terrible vomiting". Full time and weighed 9 lbs. at birth. Breast fed. Was gaining weight till 4th week. At the beginning of the 5th week vomiting commenced, and weight was rapidly lost. The vomiting was projectile, sometimes being forced down the nostrils, and it was "retentive" in type. Bowels normal before vomiting began, but since then have been very constipated. Phimosis present. The baby is small and badly nourished. Peristaltic waves can be seen passing across the abdomen, starting below the left costal margin and passing as low as the umbilicus. The pylorus is not palpable. Weight = 8 lbs. 10 oz. Was treated by careful dieting and daily gastric lavage for 6 days. A Rammstedt operation was then performed by Mr Fraser, who found that the Pylorus was/

Weight Chart of

Case 18.

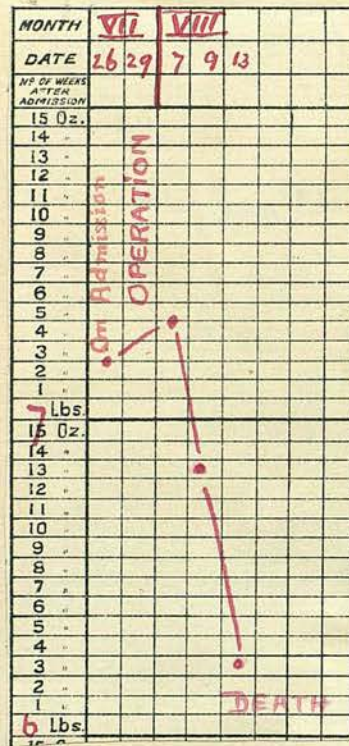
was dense and hard and enlarged to about the thickness of a lead pencil. The serous and muscular coats of the pylorus were slit parallel with the longitudinal axis of the bowel, and the mucous coat was seen bulging into the wound. Care was taken that the pylorus was incised right up to its junction with the duodenum. The pylorus was then replaced and the abdominal incision closed by through and through sutures of silk worm gut. Seven days later it was found that the abdominal wound was healing very slowly. The child's condition was very low, and green slimy stools were being passed frequently. Seven days after the operation death occurred. It was found Post Mortem that no inflammation nor sepsis of any kind had occurred. The operation in itself had been a complete success and the previously tightly constricted pyloric ring, as the result of the incision, had widened into a U-shape, ~~and~~ through the limbs of which bulged the mucous membrane. A transverse section showed microscopically the typical hypertrophied condition of the inner muscular layer, which forms the constricting circular fibres of the Pylorus. (See Plates 3 & 4)

Death in this case was due to Broncho-pneumonia

CASE 18. (See Plate 2 - page)

Male, aged 6 weeks. This is the second child, family history negative. Slight asphyxia at birth. Full time healthy looking baby. Weight at birth =

Weight Chart of
Case 22.

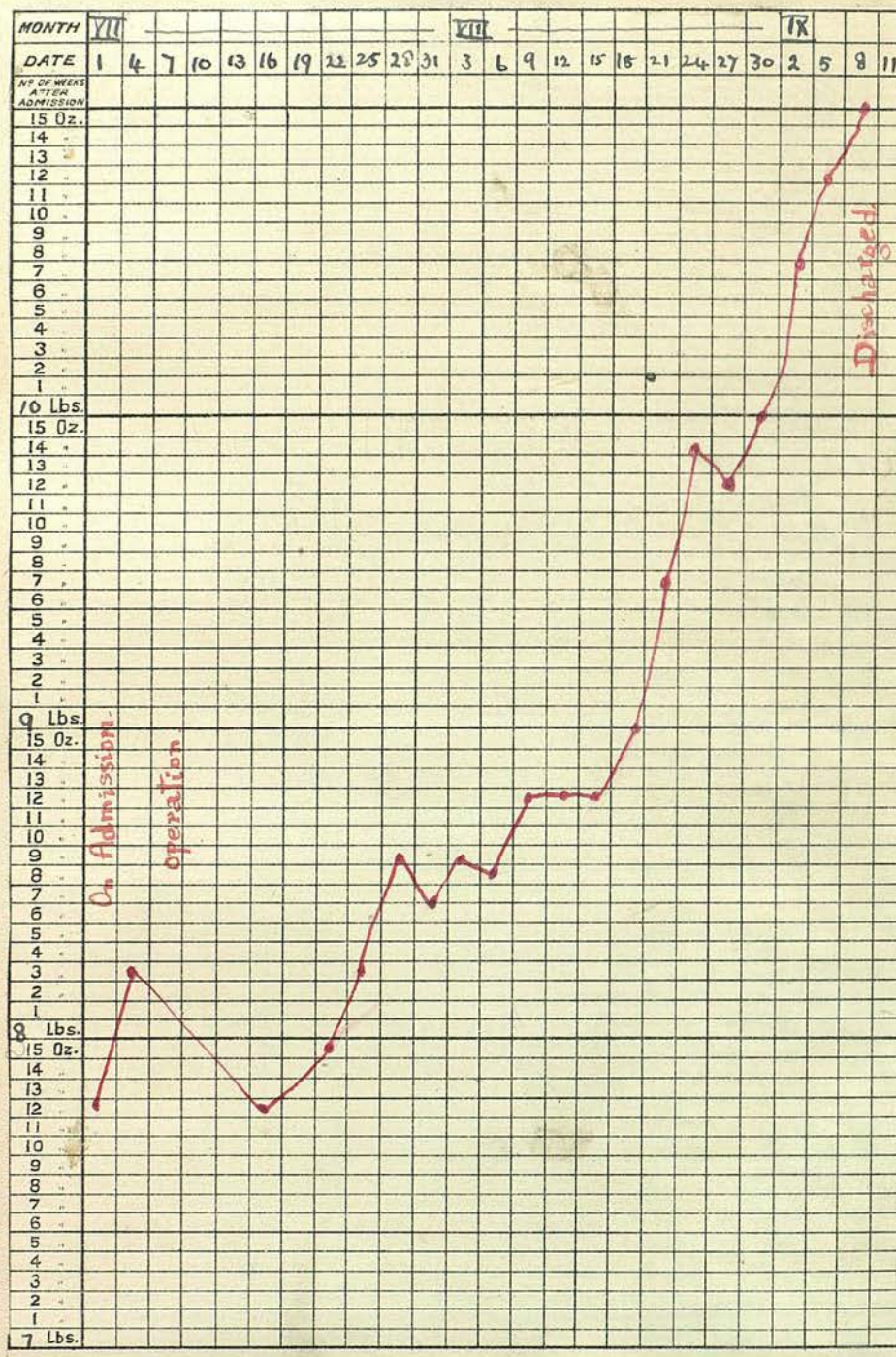


9 lbs. Has never had the breast. Cow's milk feeds have been given carefully and regularly. At 2 weeks of age vomiting began, and his feed was therefore changed several times, but with no beneficial result. The vomit was described as being "lashed up", and practically everything was brought up a few minutes after each feed. The motions were loose, very green and foul. Slightly projectile vomiting occurred during the examination. He was given a feed and very definite Peristalsis was then seen, 2 or 3 waves at a time. Present weight = $6\frac{1}{2}$ lbs. Was given peptonized milk feeds and a daily stomach wash out. For a month he received this treatment but, although no great loss of weight had occurred, things still remained stationary. An operation was therefore decided upon and Mr Fraser performed a Rammstedt. The pylorus was found to be markedly hypertrophied (See Plate 2). The shock of the operation was apparently too great as death occurred 12 hours later.

CASE 22.

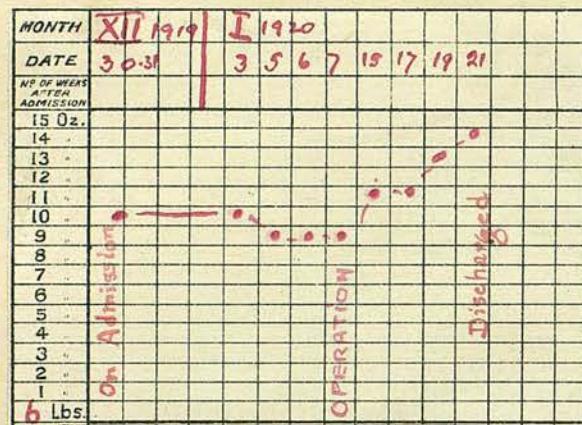
Female, aged 12 weeks. She is the second child. She is said to have vomited ever since birth. Was a very healthy looking child at birth and weighed $10\frac{1}{2}$ lbs. Breast fed at first and appeared to be always hungry, but after nearly every feed she vomited. Sometimes the milk would be vomited uncurdled, /

Weight Chart of

Case 13.

is losing weight. Has a well marked Phimosiis and a Right Inguinal hernia. Child is thin, wasted, and pale and the eyes are sunken. No depression of the fontanelle. Peristalsis is very well marked after feeds. During the night he vomited violently and looked very collapsed. Requires stimulants and is having subcutaneous salines. Daily stomach wash out was started as soon as child was in a fit condition. A week after admission the general condition had been somewhat improved and a ~~Ram~~mmstedt operation was performed. The constriction was firm and the hypertrophied pylorus was about the thickness of a "fountain pen". The recovery proved very slow and many times he was so collapsed that the outlook seemed well nigh hopeless. In spite of a gradual improvement in general condition for about 3 months there was still a little vomiting occasionally. He was circumcised about 3 weeks before his discharge, and ^{during} ~~in~~ that time there does seem to have been less vomiting, which is interesting in the light of the theory held by Tyrrell Gray and Pirie. This child is now a fine healthy looking boy of 2 years. His weight is now 1 stone 10 lbs. 4 oz. He is, if anything, above the average development and intelligence. He was weaned at 11 months and now takes the normal food for a child of his age, without the occurrence of even the slightest symptoms of gastric disturbance.

Weight Chart of

Case 17.

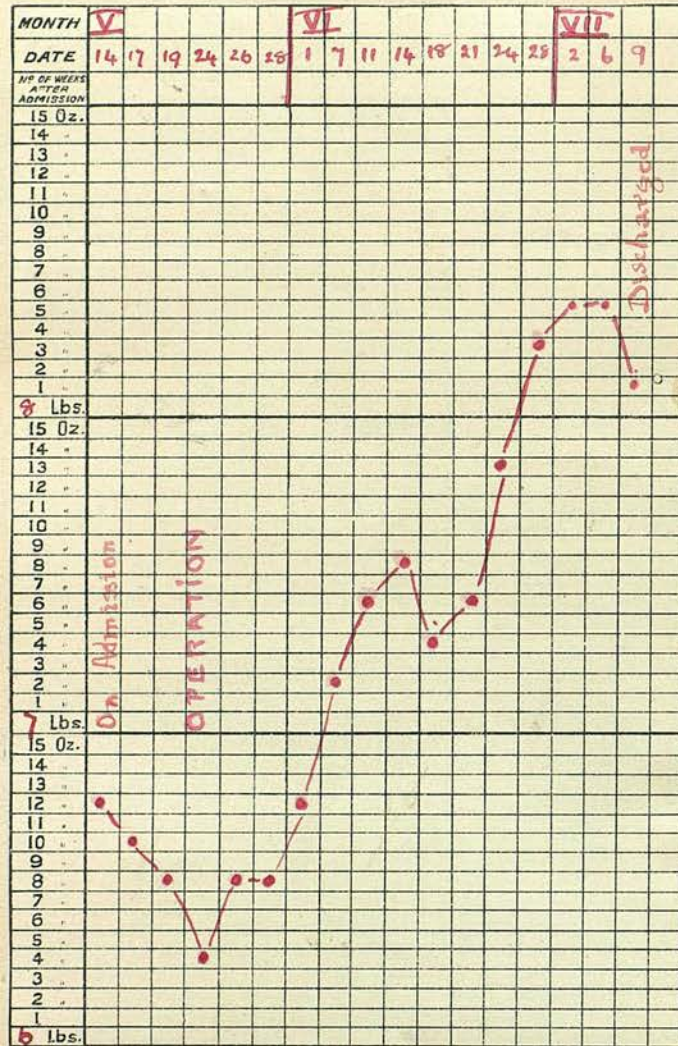
CASE 17.

Male, aged 4 weeks. This is the first child. He was a full time healthy baby and is on the breast. Has vomited since birth after nearly every feed, a slimy, curdled vomit which is usually ejected with considerable force. The bowels were at first loose but are now very costive. The mother has come for advice about the baby before going to join her husband in Canada. He is a small, puny child. A well marked phimosis is present. Peristalsis was distinctly visible during a feed which was followed almost immediately by distinctly projectile vomiting. The weight is 6 lbs 10 oz. at present. Breast feeding was continued and an occasional small enema given. The symptoms were becoming worse so a Rammstedt operation was performed a week after the child had first been seen, and a typical condition of pyloric hypertrophy was found. Peptonized milk feeds were given at first and then on the 3rd day the child was nursed by the mother, who leaned over the cot so that the infant need be disturbed as little as possible. In this case a practically uninterrupted recovery took place, perhaps owing to the fact that breast feeding was possible for the after-treatment. Since their return to Canada this child has been lost sight of, but when last seen the recovery seemed to be proving complete.

108a,

Weight Chart of

Case 20.



CASE 20.

Male, aged 5 weeks. Full time baby, was never on the breast. He appeared healthy at birth but began to vomit almost immediately and has been steadily losing weight ever since then. He has always been very constipated. On admission he was much emaciated, with a slightly depressed fontanelle. The abdomen was noticed to be prominent above the umbilicus, while below it appeared to be flattened. A stomach wash out was given daily but as the vomiting still continued it was decided that an operation was necessary. A well marked hypertrophied Pylorus was found and Mr Fraser performed a Rammstedt operation. This child was at first very collapsed but gradually rallied. For a fortnight the progress was good, then things became so stationary that it was decided to try what benefit might occur by sending the child home. In this particular case the plan worked very well and an apparently complete recovery has now occurred. The child is now 5 months old and when seen by me on the 10th of this month weighed 12 lbs. 2 oz. There has been no return of symptoms of any kind and he seems to be developing into a normal healthy baby.

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Notes on the Illustrations.Plate 1. (See page 113a.)

This illustration represents the condition that was found post mortem in the stomach of the 19th case in my series (See page 97). As has already been noted this infant had the typical symptoms and signs of Congenital Hypertrophic Stenosis of the Pylorus. He was in extremis when first seen, and although the condition was correctly diagnosed, an immediate operation was considered inadvisable as the child was practically moribund on admission.

Typical gastric peristalsis was sometimes visible after a feed, and occasionally the enlarged Pylorus could be palpated. Subcutaneous Salines were required at first, and in spite of a temporary improvement, death occurred 9 days after admission.

The post mortem examination revealed no abnormality in any organ except in the stomach. Here there was evidence of a well marked Pyloric Stenosis, the Pylorus consisting of greatly hypertrophied muscular walls about $\frac{1}{2}$ inch long. The completeness of the obstruction was proved by the following experiment:-
After /

After the stomach had been filled with water the cardiac orifice was clamped. In spite of considerable pressure it was then found that not a drop of water could be forced through the stenosed Pylorus. The naked eye appearance of the Pylorus, when cut across, suggested that the increase in thickness was due to an increase of muscular tissue. This fact was proved microscopically and it was found that the hypertrophy was principally due to a tremendous overgrowth of the circular muscle fibres. A slight increase in the interstitial fibrous tissue was present, but was probably only secondary to the muscular hypertrophy. There was no increase in thickness of the mucous membrane, but the tightly contracted state of the circular musculature caused the folds of mucous membrane to be tightly crushed together, so as almost to obliterate the pyloric lumen. These points are well brought out in the accompanying plate. It will also be noticed that the muscular coat of the stomach wall had become considerably thickened as a result of its continued peristaltic contractions.

(N.B. The green staining is due to bile).

Plate 2.

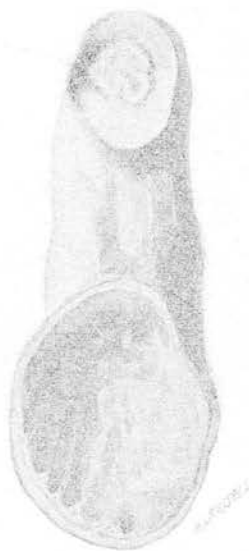


Plate 2. (See page 115a.)

The picture in this case illustrates what had taken place in a certain stomach 12 hours after a Rammstedt operation had been performed. The specimen represented by this sketch was obtained post mortem from case 18, (see page 103), death being due to the shock of the operation. The clinical features of this case were characteristic of hypertropic Stenosis, and although no tumour could ever be felt in the region of the Pylorus, the nature of the vomiting and the visible gastric peristalsis were very typical of this disease. It was at first hoped that a period of conservative treatment might enable the child to have a better chance of recovery than could be expected from immediate operation. The routine medical treatment was primarily followed by a marked subsidence of the more urgent symptoms, and by a slight gain in weight. In a fortnight, however, there was an exacerbation of the symptoms accompanied by a steady loss of weight, and it was decided that Surgery now offered the only possible chance of recovery.

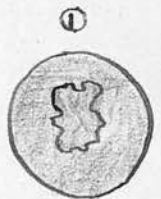
On opening the abdomen the Pylorus was found to be markedly thickened and a Rammstedt operation was therefore/

therefore performed. Unfortunately, the shock proved too great and death occurred 12 hours after the operation. Plate 2. / shows a section of the Pylorus cut transversely to the line of the Rammstedt incision, part of which is seen at the point X (see plate 2 on on page 115a). The lumen of the Pylorus, before the operation, had been practically obliterated by the crushing together of the folds of mucous membrane, resulting from the contraction and hypertrophy of the circular muscle fibres. The pre-operative condition might be represented diagrammatically thus:-

The result of cutting through a tightly contracted, hypertrophied, layer of muscle was that the divided walls immediately sprang apart like a severed elastic band, and the formerly circular organ now changed its shape into that of a U. At the operation it was noticed that the mucous membrane at once bulged outwards into the artificial gap which had been formed in the muscular sheath of the Pylorus. A diagrammatic representation of this might explain more simply what actually had occurred:-

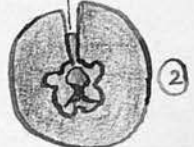
This gap in the muscle wall had formed a kind of safety valve for the relief of pressure, and the result of the bulging through it of the mucous layer was that the lumen of the pylorus had thus been rendered patent. This fact is well shown in Plate 2. (See page 115a), and it is also shown how the incision had become filled with haemorrhage from the small vessels in/

Blue = muscle
Red = mucous membrane
Green = Pyloric Lumen



To represent transverse section of an hypertrophied Pylorus

Fold of mucous membrane bulging into incision in muscle



To represent section of Pylorus a few hours after Rammstedt operation had been performed.

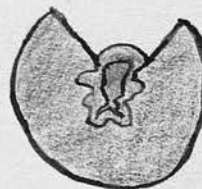
N.B. The green represents the widened Pyloric lumen.

in the Pyloric muscle that had been cut. In this case too there was present a marked thickening of the stomach. No signs of adhesion nor of inflammation were found at the autopsy.



Plate 3. (See page 118a)

In this case, the 16th of my series, death resulted from Bronchopneumonia 10 days after a Rammstedt operation had been performed. The history and clinical features of this case (see page 102.) had been typical of Congenital Hypertrophic Stenosis of the Pylorus, and at the operation the pylorus was found to be dense, hard and greatly hypertrophied. The results of the operation were entirely satisfactory, but unfortunately, as is so apt to occur, this weakly infant succumbed to an entirely different disease, that of Bronchopneumonia. It was very interesting at the post mortem examination to see the condition of affairs in the Pylorus 10 days after what we may quite justly look upon, per se, as a successful Rammstedt operation. One might show diagrammatically how the redundant fold of mucous membrane, which at the time of the operation bulged through the incision, had eventually become flattened and stretched out by the gradually separating arms of the now U-shaped layer of circular muscle. The pyloric lumen had now become quite definitely patent. The illustration on plate 3 to which these notes refer, is a very accurate representation of the post-mortem appearance/



③

N.B. This diagram shows by the increase in size of the green area, how the pyloric lumen has now become patent.

appearance of this pylorus. There were no signs of adhesions nor of any inflammation, but covering the surface of exposed mucous membrane was a thin film of fibrous tissue. This was probably the only remnant of a blood clot similar to that which was found filling the incision in the specimen represented by Plate 2. (See page 115a.)

Again in this case it was observed that there was present a very definite hypertrophy of the gastric wall as compared with that of a normal stomach.

120a.

Plate 4.



Plate 4. (See page 120a.)

A side view of the same specimen as was represented by plate 3 (page 118a.) is shown by this illustration. The advantage of viewing the Pylorus from the side is that one thus gains some idea of how wide the incision may gape, owing to the remarkable elasticity of the muscle fibres. The very great thickness of the muscular coat of the pylorus is well demonstrated in plate 4, if one examines the right hand wall of the incision.

So widely had the lips of the wound separated that one would think, on looking at the post mortem specimen, that a wedge of muscle had been removed from the wall of the pylorus. The floor of this gaping incision is formed by the outer surface of mucous membrane, which connects the two limbs of the U, formed by the circular muscle that had been severed. The accompanying illustrations are a faithful representation of the post mortem appearances that were found in the stomachs of some of the cases that have been previously described.

C O N C L U S I O N .

In conclusion, I should like to state my firm conviction that in the future, even among hospital patients, the mortality of Congenital Pyloric stenosis cases will be slowly but surely reduced by a more general use of Rammstedts' method of operation. The few recoveries after operation that have occurred in this hospital, under most adverse conditions, and the unquestionable relief from obstructive symptoms that has followed in certain cases where death from some other cause has eventually occurred, make one look forward hopefully to the future treatment and prognosis of this disease.

It gives me great pleasure to acknowledge my indebtedness to Dr John Thomson for his invaluable help. I am also deeply indebted to the Honorary Physicians and Surgeon of the Royal Hospital for Sick Children for their permission to publish notes on certain cases of Congenital Hypertrophic Stenosis of the Pylorus that have been under their care; and for facilities granted by them for me to study at the Libraries of the Royal College of Physicians, and that/